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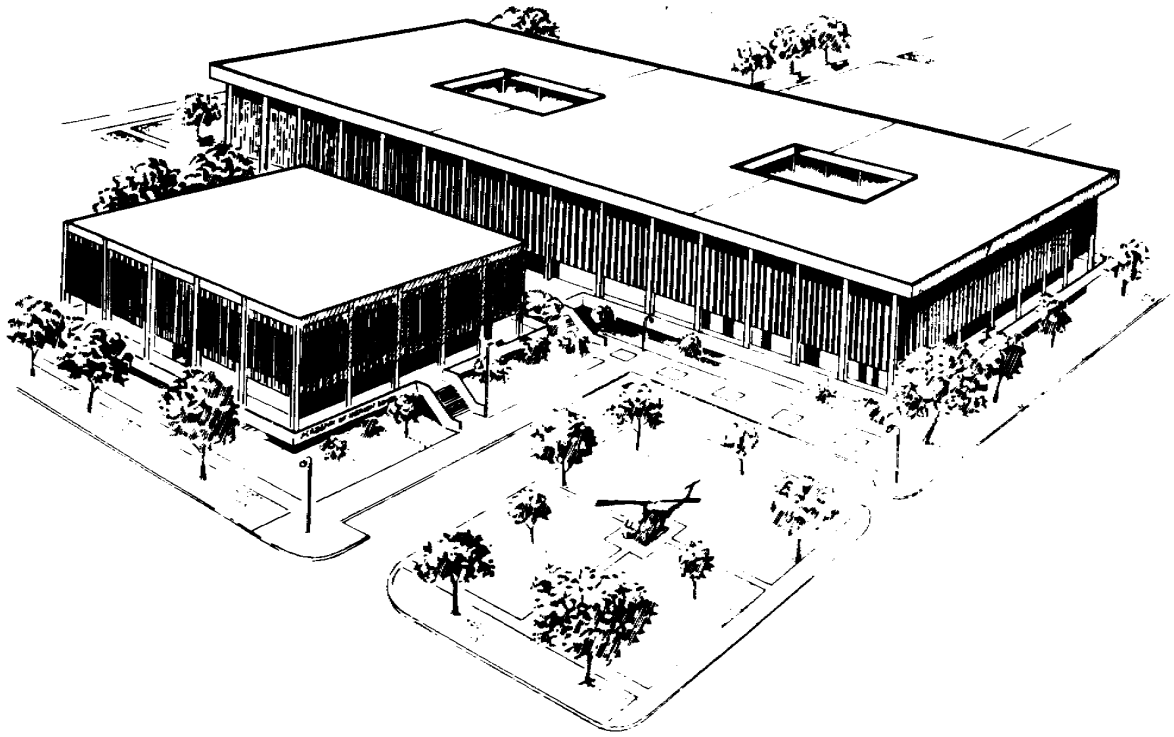
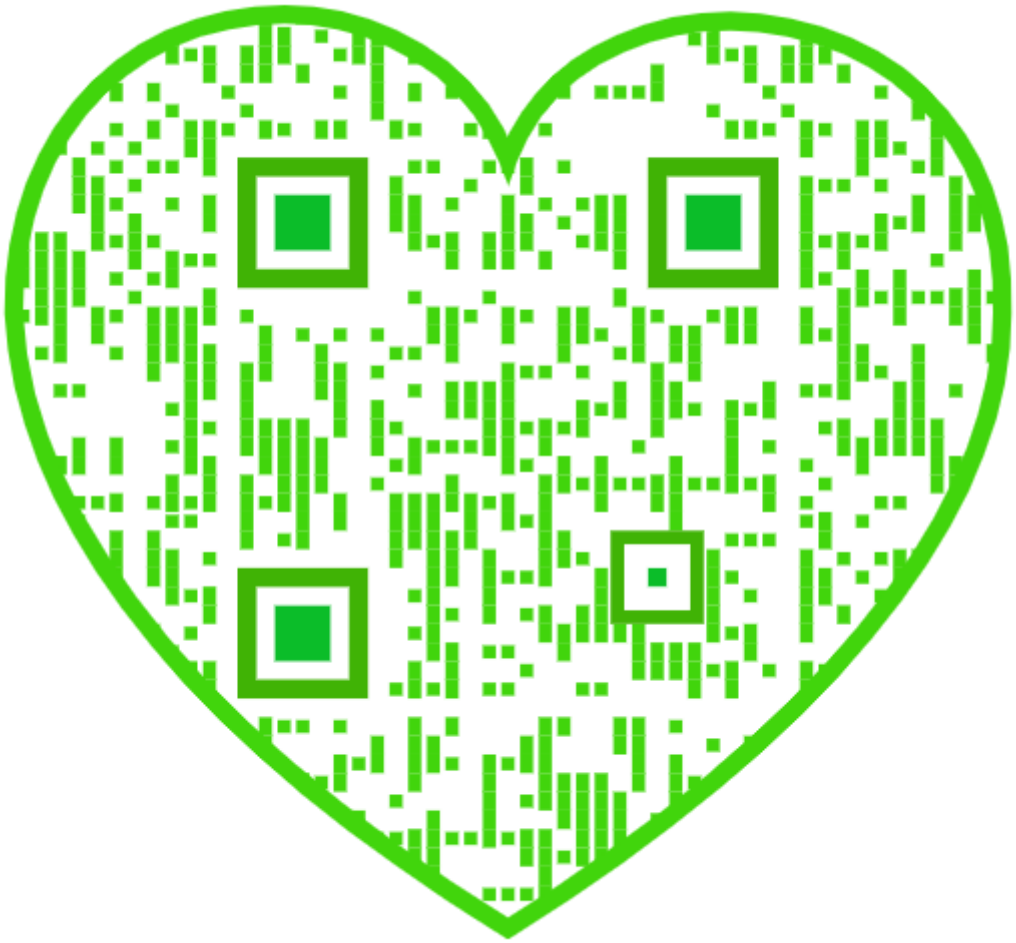


ORAL AND MAXILLOFACIAL PATHOLOGY

SUBCOURSE MD0511 **EDITION 200**



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CORRESPONDENCE COURSE OF THE
U.S. ARMY MEDICAL DEPARTMENT CENTER AND SCHOOL

SUBCOURSE MD0511

ORAL AND MAXILLOFACIAL PATHOLOGY

INTRODUCTION

Man's survival is constantly threatened--by bacteria, by physical and chemical injury, and by a host of other agents. Although modern science and medicine have done much to cope with these harmful influences, our greatest protection is still afforded by the ability of our bodies to withstand these repeated attacks. If the combination of science and natural resistance is unable to ward off the damaging influences, disease will result. The study of disease and of alterations in function caused by disease is called pathology. Frequently considered in this field are such topics as the cause of the disease, the effect of the disease on the body, and the body's reaction to the disease process.

This subcourse is designed to acquaint you with the fundamental concepts of oral and maxillofacial pathology. It also seeks to familiarize you with the technical nomenclature used in pathology, which is essential to properly understanding dental procedures. It also stresses the cause and effect of oral infection.

Subcourse Components:

This subcourse consists of three lessons. The lessons are as follows:

Lesson 1, Diseases of the Hard Tissue.
Lesson 2, Diseases of the Soft Tissue.
Lesson 3, Fractures and Dislocation of the Jaw.

Credit Awarded:

To receive credit hours, you must be officially enrolled and complete an examination furnished by the Nonresident Instruction Section at Fort Sam Houston, Texas. Upon successful completion of the examination for this subcourse, you will be awarded 7 credit hours.

You can enroll by going to the web site http://atrrs.army.mil and enrolling under "Self Development" (School Code 555).

A listing of correspondence courses and subcourses available through the Nonresident Instruction Section is found in Chapter 4 of DA Pamphlet 350-59, Army Correspondence Course Program Catalog. The DA PAM is available at the following website: http://www.usapa.army.mil/pdffiles/p350-59.pdf.

LESSON ASSIGNMENT

LESSON 1	Diseases of the Hard Tissue.
LESSON ASSIGNMENT	Paragraphs 1-1 through 1-59.
LESSON OBJECTIVES	After completing this lesson, you should be able to:

1-1. Identify terminology related to tissue inflammation of the oral cavity.

1-2. Identify the results of oral tissue inflammation.

1-3. Identify tooth structure problems caused by chemical, mechanical, or other means

1-4. Identify the results of dental caries.

1-5. Identify major diseases of the dental pulp.

1-6. Identify major diseases of the periapical tissue.

1-7. Identify major periodontal diseases.

1-8. Identify major abnormalities of the oral mucosa.

SUGGESTION After studying the assignment, complete the exercises at the end of this lesson. These exercises will help you to achieve the lesson objectives.

LESSON 1

DISEASES OF THE HARD TISSUE

Section I. GENERAL INFORMATION ON ORAL PATHOLOGY

1-1. GENERAL

a. Pathology is the study of disease, especially of the structural and functional changes in tissues and organs that cause or are caused by disease. Oral pathology is that branch of pathology concerned with diseases of the oral cavity. Microbiology is the study of microorganisms, including bacteria, fungi, and viruses. Disease is a particular destructive process in an organism, usually with a specific cause and with characteristic signs and symptoms. A sign is objective evidence of disease that is detectable by the health care provider; for example, redness or bleeding. A symptom is subjective evidence of disease--in other words, what a patient tells the health care provider. Examples of symptoms would be pain and weakness. The word asymptomatic refers to a disease exhibiting no symptoms.

b. Terminology of a descriptive nature is used to define aspects of a pathological process. Local (localized) means that the disease is restricted to a part or small area of the body. Generalized means the disease affects all parts of the body or covers a large area (many organ systems). Incipient refers to a disease or disorder that is just beginning. Severe refers to an intense distress of prolonged duration caused by the disease process. Acute relates to a sudden onset of symptoms, generally of short duration. Chronic refers to a prolonged disease process, with symptoms generally of a constant nature.

NOTE: As you study this subcourse, you may wish to refer to Subcourse MD0501, Dental Anatomy and Physiology.

1-2. INFLAMMATION OF TISSUES

When a tissue is injured or irritated by mechanical, chemical, thermal, or bacterial agents, it becomes inflamed. Inflammation is the reaction of a tissue to an injurious agent. The body does not always overcome the attack and so disease persists. For example, the irritation produced by dental caries may result in inflammation of the pulp. This pulpal inflammation is called pulpitis. (The suffix "itis" indicates that inflammation exists). In every case, the inflammatory condition results from some form of irritation and, in order to restore normal function to the tissues, the cause must be eliminated. The function of inflammation is to activate all the defenses of the body and to bring them to the site of the injury with the purpose of overwhelming the source of the injury or irritation.

1-3. STAGES OF INFLAMMATION

Inflammation is a body defense mechanism that occurs regardless of the source of injury. The inflammatory response is progressive, in stages; however, it should be realized that various stages may exist within an injured area at the same time. The major stages in the inflammatory process are vascular change, exudation, and repair. The initial inflammatory response is vascular tissue injury resulting in the following sequence of events.

a. **Hyperemia.** The presence of an abnormally large blood supply in which the blood vessels are dilated and the flow of blood is slower.

b. **Permeability.** Increased permeability in the smaller blood vessels, allowing the body to position blood elements in the area in order to heal the injured tissue or combat any introduced foreign agents.

c. **Exudation.** The movement of fluid and blood cellular elements (exudates) into the injured tissue(s).

d. **Edema.** Accumulation of fluids within the tissue(s) that then slows or stagnates blood flow. This acts to localize the inflammatory process.

1-4. MAJOR SIGNS AND SYMPTOMS OF INFLAMMATION

The four major signs and symptoms of inflammation are swelling, pain, heat, and redness. Sometimes a fifth sign is disturbance of function. Swelling is the visual sign of the accumulation of inflammatory substances (exudates) in the tissues and the increased amount of blood in the area. The pressure of the exudates (and perhaps the action of toxins, enzymes, and acids released from injured cells or liberated by defensive cellular elements) on the nerve endings causes pain. The redness and heat that accompany inflammation are because of the increased amount of blood in the area. Inflammatory involvement of the tissue may result in disturbance of function.

1-5. BENEFICIAL EFFECTS OF INFLAMMATION

The beneficial effects of inflammation are essentially fourfold. First, the increased fluid dilutes the irritants present in the area (e.g., bacteria and their poisonous products). Second, the blood cells engulf and often digest bacteria, dead cells, or other debris that might cause or continue the inflammation. Third, antibodies, which are also present in the edema fluid, neutralize toxic substances. Fourth, clotting of the edema fluid walls off the area and prevents the irritant and the inflammation or the inflammatory process from spreading beyond the affected site.

1-6. SUPPURATION

Suppuration is the formation of pus. It often accompanies inflammation and retards the healing process. Pus is composed of leukocytes, broken-down cells, serum, bacteria (dead or alive), and inflammatory debris.

1-7. ABSCESS

An abscess is a circumscribed area of pus surrounded by a restraining wall of tissue. A narrow tube or channel that often develops to afford drainage of the abscess is called a sinus track. A periapical abscess is a collection of pus at the apex of the tooth. It is caused by the spread of infection through the apical foramen of the tooth to the periapical tissues. A periodontal abscess is a collection of pus along the sides of the tooth but usually not involving the apical area. It may be caused by a foreign object forced into the periodontal tissues. Abscesses are often clinically manifested by swelling.

1-8. ULCER

An ulcer is an open sore other than a wound. The base of an ulcer is composed of granulation tissue (wound repair tissue) resulting from the body's attempt at healing and repair.

1-9. CELLULITIS

Cellulitis occurs when inflammation is not controlled and contained within a localized area and spreads through the substance of the tissue or organ. In cellulitis, swelling usually develops rapidly in conjunction with a high fever. The skin usually becomes very red and the area is characterized by severe throbbing pain as the inflammation localizes. The condition is often associated with periapical, periodontal, or pericoronal infections.

1-10. REPAIR OF TISSUES

a. **General.** The capacity of the human body to restore damaged tissues to health varies with the particular type of tissue or cells involved. Worn-out cells are replaced and tissues rebuilt as a normal physiological process to bring about the growth and repair of the body as a whole. Once tissue injury has been controlled by the inflammatory process, wound repair can begin. Cells in the area of the injury proliferate, organizing to return the tissue to its original form with resumption of previous function. The process of healing is fundamentally the same in all damaged tissues. It consists of two parts. First is the removal of inflammatory material and necrotic debris. The second part, as much as possible, is the replacement or reconstruction of the original tissue.

b. **Granulation.** The process of healing involves the invasion and replacement of dying and dead tissue by immature mesenchyma called granulation tissue. Granulation tissue consists mostly of fibroblasts and capillaries. As wound repair progresses, the fibrous and vascular cell components gradually convert into a mature tissue. Then, old epithelial cells at the wound edge proliferate to cover the new fibrous tissue surface. If the wound edges are not brought together or are exposed to irritants or infections, granulation tissue forms on the opposing surfaces and fills the space.

c. **First Intention.** This is the healing process observed when the wound surfaces are close together, such as in a small cut or in a closely sutured wound. The first step in repair is the formation of a clot. This clot is slowly replaced by granulation tissue and a covering of epithelium. When the edges of a clean aseptic wound are closely approximated, rapidly growing (proliferative) connective tissue cells join the walls of the wound and proliferative epithelial cells close over the surface. In first intention, the wound heals without scarring.

d. **Second Intention.** This is the healing process observed when the opposing edges of the wound are not together, such as in a large ulcer. Granulation tissue fills in the wound from the base and eventually is covered with epithelium. In many instances, repair results in less than completely normal regeneration, causing altered tissue structure that forms a fibrous scar. In second intention, healing is often characterized by the formation of a scar.

Section II. EROSION, ABRASION, AND RESORPTION

1-11. GENERAL

There are conditions (besides dental caries) in which there is loss of tooth substance. They are erosion, abrasion and attrition, and resorption.

1-12. EROSION

Erosion is the chemical wearing away of the tooth structure by a chemical process that does not involve bacterial function. A common cause of erosion is chronic vomiting which results in enamel destruction through the action on teeth by stomach acids. Excess use of acidic carbonated beverages or acid citrus fruits may also be causative factors. Erosion is usually found on the external surface of the tooth on enamel structure adjacent to the cemento-enamel junction. It appears as various shapes cut into the surface at the neck of the tooth. The enamel and dentin are usually hard and shiny. In certain cases, the crowns may be severed from the roots. Considerable variation in appearance is normal between cases, but generally several teeth are involved, usually on the outer (labial) aspect of the crown.

1-13. ABRASION AND ATTRITION

Abrasion is considered to be an abnormal or excessive wearing away of tooth substance by a mechanical process. It can be seen clinically. In abrasion, one or more teeth may show the effects of wear. The wearing of the tooth substance may be caused by biting some foreign substance such as a pipe stem or a bobby pin, by faulty tooth brushing techniques, or by nervous biting habits. Attrition is the wearing away of tooth structure because of contact with an opposing tooth. It involves all the teeth with the cusps and contact points of all the teeth showing uniform wear. Abrasion and attrition differ in that attrition is considered a normal, gradual loss of tooth substance from the chewing of food and it involves all of the teeth, which will show uniform wear. If the loss of tooth substance is excessive, it is called abrasion. In both cases, the dentinal tubules may become calcified and secondary or irregular dentin may be deposited in the pulp immediately below them. Secondary cementum may be laid down about the roots.

1-14. RESORPTION

a. **General.** In resorption, there is the dissolving (removal) of body tissues by body fluids or cellular activity. Osteoclasts (cells that destroy bone) and cementoclasts (cells that destroy cementum) are the cells active in the resorption of bone and cementum. The condition is evident most often in the alveolar process and in the roots of teeth. Certain types of resorption may be considered normal. Other types may be the result of abnormal conditions. Healthy bone is constantly being remodeled. Resorption and deposition of bone is a normal physiological process and the principle that allows orthodontic movement of teeth. Stimulation of resorptive cellular activity through excessive mechanical or occlusal forces or through the normal shedding of the primary dentition is also within normal resorptive function. The effects of resorption can often be seen on a radiograph. Pathological resorption may be initiated through inflammatory stimulation or as the result of pressure exerted by developing neoplasms or cysts.

b. **Normal Resorption.** The roots of deciduous teeth are resorbed just before the eruption of the permanent teeth. There is also normal resorption of bone in edentulous areas where permanent teeth have been extracted. Healthy bone is constantly being remodeled. Resorption and deposition of bone is the basis for the treatment applied by the orthodontist who moves malposed teeth slowly into proper position. Resorption is also important in the repair of a fracture and the healing of a tooth socket.

c. **Abnormal Resorption.** There are other types of resorption that may not be considered normal. Resorption of the roots of permanent teeth may be the result of many factors involving disease and trauma. For example, systemic (endocrine) disturbances and some neoplasms are characterized by bone and root resorption. Impacted teeth may impose upon the roots of other teeth to cause areas of resorption.

d. **External and Internal Resorption.** Resorption of a tooth may develop externally or internally. Internal resorption is usually caused by inflammatory pulpal stimulation. External resorption may be caused by inflammatory stimulation of resorptive cells or through stimulation of resorptive cells associated with the pressure exerted by developing pathology or impacted teeth. This can cause resorption of tooth structure and surrounding alveolar bone. The cell responsible for the active resorptive process in bone is an osteoclast just as the cementoclast is for cementum. The osteoclast is developed from connective tissue cellular components in the pulp or area adjacent to an inflammatory stimulus outside the tooth.

Section III. DENTAL CARIES

1-15. GENERAL

Dental caries is a term for the process commonly referred to as decay of the teeth. Thus, a decayed tooth is properly termed a carious tooth. Dental caries or dental decay is a specific disease that causes dissolution and disintegration of the hard structures of the tooth, that is, enamel, cementum and dentin. (The word caries or decay refers to the disease; the word cavity refers to the lesion or hole in the tooth.) Dental caries is the most widespread disease affecting the human race. The incidence of caries is greatest during childhood and young adulthood. It attacks deciduous teeth the same as it attacks permanent teeth. Dental caries is progressively destructive. It usually begins in a minute area on the enamel or cementum and, if untreated, progresses to the dentin. The next step is penetration to involve the dental pulp. Infection and death of the pulp may follow, with possible extension of the infection into the tissues surrounding the apical portion of the root and the formation of an abscess. The control of dental caries is a very important problem that is receiving much attention in the fields of research and prevention.

1-16. CAUSES

In general, dental caries occurs because of improper or poor oral hygiene. The destruction of dental tissue by caries, however, is governed somewhat by the susceptibility of the teeth. Little is known about susceptibility or resistance to caries, but the degree of susceptibility may be influenced by certain factors including diet, oral hygiene, and some systemic diseases. Study of the direct cause of dental caries is very complex. Only two theories of its cause are considered here.

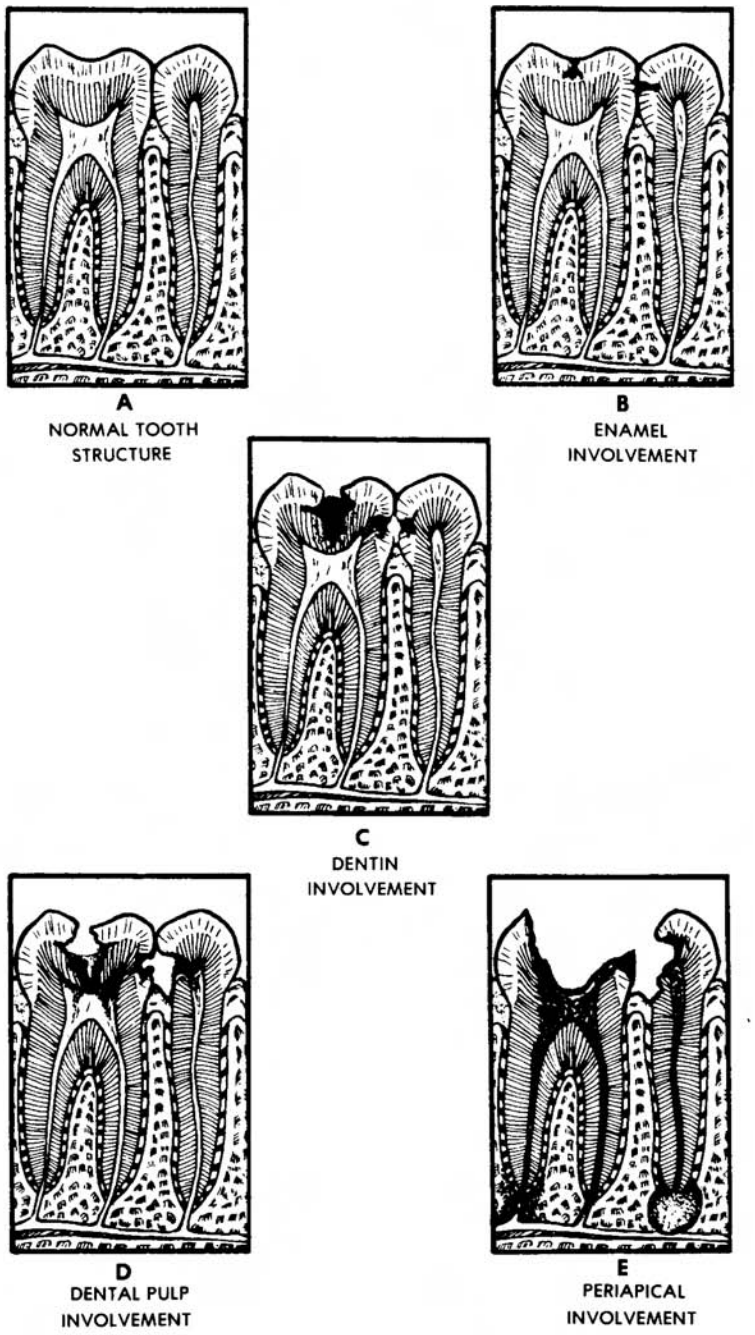
a. **Acidogenic Concept**. According to the acidogenic (producing acid) concept, bacteria and their products accumulate in mucinous plaques, which are often invisible and adhere tightly to the teeth. The bacteria in the plaques metabolize (feed upon) carbohydrates in the diet and convert them to organic acids. These acids dissolve the enamel and allow deeper bacterial penetration into the tooth. The lesion is progressive.

b. **Proteolytic Concept**. According to the proteolytic (effecting the digestion of proteins) concept, proteolytic bacteria attack the organic material in the enamel rods (which is mostly protein) along certain tracts called lamellae. Dissolution of the organic material in these lamella tracts eventually allows bacterial penetration to the underlying dentin.

c. **Combined Concepts**. According to both concepts, dental caries is the result of microbial activity on the teeth. The process of dental caries may be a combination of both concepts, with the acidogenic concept accounting for dissolution of mineral content and the proteolytic concept accounting for destruction of organic content of the tooth.

1-17. CARIES IN ENAMEL

The organisms that produce acid are contained in mucinous plaques that adhere to the surface of the enamel. Common sites of plaque adherence are pits, fissures, interproximal areas, and along the free margin of the gingiva, particularly on the facial surface of the tooth. The enamel rods of a tooth are cemented together by a substance that dissolves more readily than the rods themselves and, thus, according to the acidogenic concept, the first effect in enamel caries is probably the dissolution of this cementing substance. The first visible evidence of caries is a slightly whitened area on the surface of the tooth (decalcification). This area is very easily overlooked, particularly when the teeth are wet. Drying of the tooth surface makes these areas far more visible. From the small areas on the surface of the tooth, caries continues its progressive destruction. As the acid dissolves the cementing substances, the enamel rods are left without support and break away. Since enamel contains a minimal amount of organic material, the primary acid effect on the enamel cementing substance results in loss of enamel structural support. Without support, the enamel rods break away, allowing progressive formation of a cavity within the enamel, until ultimately dentin is also involved. In summary, caries first penetrates the enamel and dentinoenamel junction. Then it spreads laterally and deeper within the dentin toward the pulp. Thus, what may appear to the patient as a small surface cavity may in fact be a very extensive involvement of the tooth structure. See figure 1-1.



NOTE: Dental decay generally starts in areas that are not easily or normally cleaned. Figure B shows primary areas of enamel decay, between the teeth and on the chewing surface. Spread of decay in dentin is shown in figure C; note lateral advancement as well as progression of decay toward the pulp. With pulpal involvement, degenerative changes result in pathology within the pulp chamber (figure D), resulting in eventual periapical granuloma or cyst development (figure E).

Figure 1-1. Stages in the progress of dental caries.

1-18. CARIES IN DENTIN

From the small area on the surface of the tooth, caries continues its progressive destruction. As noted in the preceding paragraph, decay spreads easily within dentin after penetrating the enamel. The disease spreads laterally along the dentinoenamel junction and directly toward the pulp.

a. **The Destructive Process.** The destructive process of caries differs in dentin and enamel. In caries of enamel, the organisms cannot enter the substance of the enamel until the acid produced by the organisms has destroyed the enamel substance. This process is called demineralization. The reverse is true of the dentin, which is made of many hollow tubules. These tubules offer a natural pathway for penetration by bacteria and because the dentin contains a large proportion of organic materials, progression is faster in dentin than in enamel. When the dentin has been reached, acid-forming and proteolytic organisms can enter the dentinal tubules and produce acid or break down the organic matrix within the tissue itself. Since dentinal tubules tend to branch and communicate with each other at the dentinoenamel junction, the organisms penetrate the dentin laterally in all directions along this junction. Accompanying this lateral penetration is a penetration along the main tubules in the direction of the pulp. This lateral and direct involvement tends to form a cone-shaped area of decay. The apex of the cone is pointed toward the pulp of the tooth and the base at the dentinoenamel junction.

b. **Limits to Dentin Destruction.** Although dentin destruction in caries is normally faster than enamel destruction, the rapidity of dentin destruction both in depth and breadth will be governed by its structure. Because of the extensive organic matrix of the dentin, enough substance is left after the dissolution of the inorganic salts to retain its physical form until further destruction has taken place by other processes.

c. **The Infected Layer and the Affected Layer.** Caries in the dentin is described as having an infected layer and an affected layer. In the infected dentin, many microorganisms are present and most of the dentinal tubules have been destroyed through demineralization and decomposition (destructive process of proteins). The infected layer is soft on the surface but gets tough, leathery, or rubbery underneath. Below the infected layer is the affected layer of dentin. Few microorganisms are found in this area and the dentinal tubules are intact. Normal dentin is found under the affected layer. The difference in these layers plays an important role in the treatment of caries in the dentin.

1-19. ARRESTED CARIES

Arrested caries is the stage where the progress of the decay process has stopped. The softened dentin has been lost or worn away so that the discolored (either yellow, brown, or black), sound, hard dentin remains. The remaining dentin has a polished look.

1-20. PULPAL REACTION TO DENTAL CARIES

a. **The Reason for Pain.** When dental decay reaches a depth in the dentin that is near the pulp tissue inside of the tooth, the pulp tissue can become inflamed. The inflammation causes the blood vessels to swell and release fluid into the extra cellular spaces. This swelling is limited by the hard walls of the pulp chamber and root canals and, as a result, severe pain may result because of the constriction. Pus can accumulate within the diseased pulp tissue that further accelerates the pathologic process.

b. **The Process of Decay.** The reaction of the pulp to dental caries varies. This reaction generally varies in accordance with the rate of advance of the carious process. Even before there is significant dentin involvement, the pulp tissue can become irritated and the irritation causes minor inflammation. This, in turn, stimulates the odontoblasts to produce secondary dentin in an effort to protect the pulp. If the rate of advance of caries is slow and the resistance of the patient is high, this deposition of secondary dentin may prevent the carious process from reaching the pulp tissues for a considerable time. If it were not for this protective reaction of the odontoblasts, a far higher percentage of carious lesions would reach the pulp chamber before detection, causing more injury to the pulp. If not treated or arrested, the carious process can eventually reach and involve the pulp tissue. As it nears the pulp, the irritation becomes more severe and direct bacterial invasion occurs. If the resulting inflammation is severe or of prolonged duration, the pulp tissue may die (necroses) if left untreated.

Section IV. DISEASES OF THE DENTAL PULP

1-21. GENERAL

Pulp is an extremely sensitive tissue that can only elicit pain. It can be subjected to many irritations resulting from dental caries, exposure to excessive heat or cold, and mechanical, chemical, or electrical stimulation. In a healthy tooth, the enamel and periodontal tissues act as mechanical and insulating coverings. If the enamel or cemental tissues covering the root are damaged or removed, the underlying dentin is exposed. The dentin then becomes sensitive to external irritants because of the movement of fluid in and out of the dentinal tubules. This fluid movement causes stimulation of the sensitive pain receptors inside the dentin pulp. Injuries such as dental caries, trauma, developmental defects and/or diseases, or dentistogenic (dentist-caused) injuries can have a negative effect on the pulp. Among the diseases that occur in the pulp are acute pulpitis, chronic pulpitis, pulp abscesses, hyperplastic pulpitis, and a number of degenerative changes (diseases) that take place. Acute and chronic pulpitis and pulp abscesses are commonly encountered in clinical dental practice.

1-22. ACUTE PULPITIS

Acute inflammatory changes in the pulp are often caused by bacteria that have gained access to the exposed pulp. These changes may cause an initial inflammation or a flare-up of a chronic inflammation. The active growth of the organisms in the environment of the pulp produces rapid destructive changes. These changes are accompanied by symptoms which can be correspondingly severe. Clinically, severe pulsating pain is caused by the rapid buildup of pressure within the tooth. Release of pressure by opening into the pulp chamber brings almost immediate relief. If the condition is severe enough, it may be accompanied by fever, headache, and malaise. As the inflammation progresses, it gradually may involve the entire pulp. Inflammation can spread to the periapical tissues and cause the tooth to loosen and be sensitive to externally applied pressure. It may pass beyond the periapical tissues to involve the surrounding bone and perforate the periosteum and overlying tissues to drain either into the mouth or through the skin.

1-23. CHRONIC PULPITIS

Chronic pulpitis may be the result of persistent mild to moderate irritation of the pulp or it may follow a period of acute pulpitis. Chronic pulpitis may either resolve itself or continue with slow destruction of the entire pulp, involving also the periapical tissues. Chronic pulpitis may also develop into an acute infection in the presence of virulent organisms that have reached the pulp from the mouth. Chronic pulpitis is usually characterized by intermittent periods of mild to moderate pain or no pain at all. These intermittent periods are associated with periods of increased activity and buildup of pressure in the pulp chamber and root canals.

1-24. PULP ABSCESS

Localized areas of infection, such as an abscess with pus, may develop within the pulp tissue. Abscesses may be found in teeth in which restorations have been placed or in teeth having no detectable lesion. They can produce severe, intermittent pain that may increase when the patient lies down. This sharp pain may respond to the application of cold, tapering off to a dull pulsation. The affected tooth is sometimes difficult to identify because of the occasional absence of caries or other identifiable causes. Lack of involvement of the periapical tissues also makes it difficult to identify the affected tooth. The tooth may not differ from the other teeth in its sensitivity to percussion or other externally applied pressure.

1-25. HYPERPLASTIC PULPITIS

Hyperplastic pulpitis is occasionally encountered in deciduous teeth as an enlargement of the pulp following carious destruction of the tooth crown. It is an unusually excessive reaction to inflammation. The exposed pulpal tissues extrude through a communication to the oral cavity and an excessive inflammatory reaction results. It is seen as a soft palpable mass (pulp polyp) overlying the pulp chamber, exposed within the crown portion of the tooth. There is usually no pain associated with this condition.

1-26. DEGENERATIVE CHANGES OF THE PULP

Since the pulp of the tooth is subjected to a variety of bacterial, mechanical, or chemical irritations, degenerative changes often occur within the pulp. Types of degenerative changes include calcific degeneration, pulp stones, and fibrosis. Degenerative changes seldom have any clinical significance. To a certain extent, they are normal physiological processes. They do, however, make any future endodontic treatment more difficult for the dental officer.

Section V. DISEASES OF THE PERIAPICAL TISSUE

1-27. GENERAL

After the pulp dies, the inflammatory process will frequently extend through the apical or lateral foramina into the adjacent tissues. Among the resulting diseases are acute periapical inflammation, periapical granuloma, radicular cyst, and periapical abscess. Extensions of infections originating in teeth may lead to such conditions as osteomyelitis and Ludwig's angina.

1-28. ACUTE APICAL PERIODONTITIS

Acute inflammation of the periapical tissue usually represents an extension of acute pulpal inflammation. The periapical tissues become inflamed and the tooth is usually very tender when used in mastication. No pathology is seen in the bone on a radiograph. The treatment consists of either root canal therapy or extraction.

1-29. CHRONIC APICAL PERIODONTITIS

A chronic inflammatory condition may occur and cause little or no pain. It may persist for a long time and pathology is usually seen in the bone on a radiograph. There may also be a sinus track present in the gingiva adjacent to the tooth.

1-30. ACUTE APICAL ABSCESS

The inflammation in the periapical tissues is very severe, resulting in swelling, fever, and sharp pain. An incision and drainage may be necessary to relieve the pain and drain the pus. There is generally no pathology seen in the bone on a radiograph. (This abscess is nearly always the result of a change from an existing chronic infection).

1-31. PHOENIX ABSCESS

The symptoms and treatment are the same as those for the acute apical abscess. However, pathology can be seen in the surrounding bone on the radiograph.

1-32. HISTOLOGY

a. **Periapical Granuloma.** Periapical (or radicular) granuloma is a common result of pulpitis. It is a mass of granulation tissue that forms at the apical/lateral foramen in response to chronic inflammation caused by necrosis of the pulp tissue. It is composed of capillaries, inflammatory cells, fibroblasts, collagen, and macrophages. It is this type of tissue that fills the bony defect seen on radiographs.

b. **Periapical Cyst (Radicular Cyst).** A periapical cyst (or radicular cyst) often forms from proliferation of islands of odontogenic (tooth origin) epithelium present within a radicular granuloma. The cystic lesion consists of a fluid-filled cavity that is lined with epithelium. The cyst generally develops from a long-standing periapical granuloma. See figures 1-2 and 1-3.

1-33. SIGNIFICANT CLINICAL INFECTIONS LUDWIG'S ANGINA

Ludwig's angina is a profound infection clinically characterized by a firm swelling of the floor of the mouth and elevation of the tongue. Swelling may spread into the tissues of the neck that can cause swelling of the tissues and airway obstruction that can cause death. This condition is relatively rare, but very dangerous. It is commonly accompanied by fever, pain, and serious interference with breathing. Extensions of infection from carious teeth, extraction sites, or tonsils may cause this disease. Treatment includes establishing an airway, administering antibiotics to control infection, and establishing drainage through incision. Referral to an oral and maxillofacial surgeon is usually required.

NOTE: This radiograph shows extensive destruction of bone in the periapical region of the mandibular incisor teeth. Note the extensive carious involvement of these teeth. A biopsy is necessary to determine whether a cyst or a granuloma has developed. Both have a similar appearance on a radiograph.

Figure 1-2. Periapical cyst, bone destruction.

NOTE: This cyst was removed from the root area of a patient's tooth.

Figure 1-3. Periapical cyst, excised.

Section VI. PERIODONTAL DISEASES

1-34. GENERAL

Periodontal diseases involve the periodontal tissues. When considering the causes of these diseases, both systemic and local factors must be taken into account. Periodontal diseases involve the supporting structure that maintains a tooth within the dental arch. This includes the periodontal ligament, the alveolar bone, and the gingiva. An inflammatory process or trauma generally initiates periodontal disease; however, systemic considerations can also influence the susceptibility and degree of involvement.

1-35. GINGIVITIS

Gingivitis (figures 1-4 and 1-5) is an inflammation of the gingival tissues. It is characterized by the typical signs and symptoms of inflammation--swelling (edema), redness, pain, increased heat, and, sometimes, disturbance of function. Most patients that appear to have clinically healthy gingiva also have minute areas of inflammatory activity. The inflammation is caused by the toxic substances produced by bacteria in the mucinous plaques adjacent to the gingival tissue. Direct irritation from food impaction, toothbrush bristles, or toothpicks may also cause gingival inflammation. With inflammation, the gingival tissue appears to proliferate. The inflammatory gingival response causes a swelling of the tissue caused by increased vascular activity. This increases the depth of the gingival sulcus (a furrow between the surface of the tooth and the gingiva) around the involved teeth, increasing the potential for continued and further gingival involvement. The increased depth of the sulcus allows additional debris to accumulate and more plaque to form. If this plaque is not removed, it may become calcified from the precipitation of calcium salts from the saliva. The calcified plaque is called calculus.

NOTE: Inflammation of the gingival tissues is evident at the neck of these maxillary and mandibular anterior teeth. Note the redness and bulbous engorgement (edema) of the tissue as well as the lack of normal tissue texture. Dental plaque accumulation accounts for this inflammatory response.

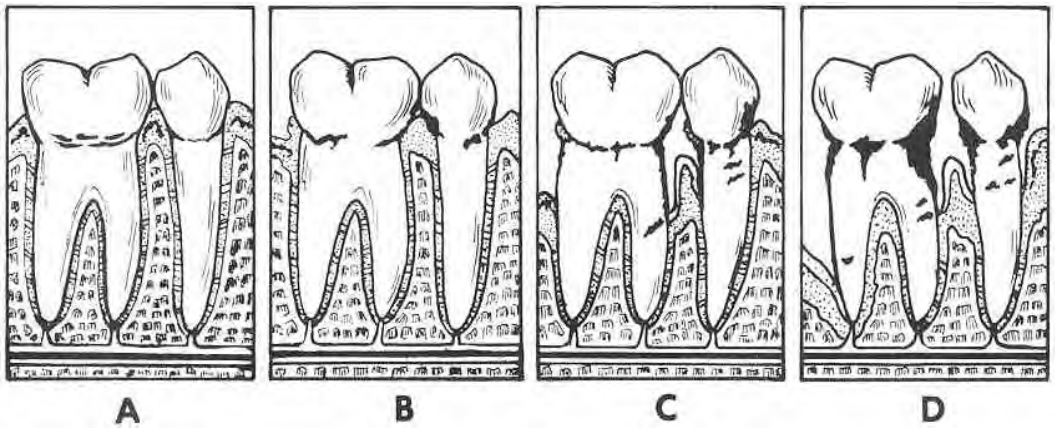
Figure 1-4. Gingivitis, tissue inflammation.

NOTE: Long term dental neglect has resulted in a progressive inflammatory response that, in some areas, has initiated a fibrous tissue development. Note areas of inflammation and fibrous gingiva. Malpositioned teeth contribute to inadequate cleansing and dental plaque accumulation).

Figure 1-5. Gingivitis, fibrous tissue development.

1-36. PERIODONTITIS

a. **General.** Periodontitis (figure 1-6) is an inflammation of the tissues supporting the teeth. It is also commonly known as "pyorrhea." If untreated, the tooth may be lost through destruction of the supporting tissues.



NOTE: Deposit of minerals from the saliva into dental plaque results in a hard calculus formation. The pockets between the teeth and gingival tissue deepen as periodontal disease progresses. Calculus formation in periodontal pockets makes plaque removal more difficult, contributing to progress of the disease and destruction of the bony support of the teeth.

Figure 1-6. The progress of periodontal destruction.

b. **Cause.** Periodontitis is caused by the local irritants of bacterial plaque. By making plaque more difficult to remove, calculus deposits, poor fitting crowns, bands, and restorations contribute to developing periodontitis. Poor diet, endocrine disturbance, and systemic disease may serve as predisposing and contributing factors.

c. **Effect.** In periodontitis, the periodontal ligament, cementum, alveolar bone, and gingival tissues are destroyed. With this loss of support, the teeth may become loose. In this disease, the periodontal ligament separates from the cementum and a pocket forms between the tooth and the gingival tissue. The pocket becomes an entrance for bacteria. Inflammation follows. Alveolar bone is resorbed and the space created is partly filled with granulation tissue. With loss of bony support, the teeth may become loose. In periodontitis, the gingival tissue may take on a bluish appearance. Bad taste, bleeding gums, and hypersensitive teeth are common symptoms. Treatment includes management of the disease by the dental officer and vigorous home care by the patient.

1-37. LOCALIZED JUVENILE PERIODONTITIS

This disease is in fact a series of diseases in teens and young adults caused by bacteria and systemic disorder. It progresses so rapidly that conventional therapy is inadequate.

1-38. PERIODONTAL ABSCESS

A periodontal abscess is most frequently a sequel of untreated periodontal disease. It is a collection of pus along the sides of the tooth that may or may not involve the apical area. It can be caused by a foreign body such as calculus, a toothpick, or popcorn hull becoming lodged in the periodontal ligament or beneath the free margin of the gingival tissue. The irritant causes an inflammatory response and, because of minimal drainage through the periodontal sulcus, pus forms. A periodontal abscess may drain either through periodontal pockets or through the gingiva into the mouth. The most common signs and symptoms are swelling, dull pain in adjacent periodontal tissues, soreness of the gingiva, and shiny mucous membrane over the area. Establishment of drainage tends to reduce the acute symptoms.

1-39. NECROTIZING ULCERATIVE GINGIVITIS (NUG)

Necrotizing ulcerative gingivitis (NUG), commonly called "trench mouth" or Vincent's disease, is a bacterial infection. It is usually associated with poor oral hygiene, smoking, and/or psychological stress, but may be seen in patients with good oral hygiene.

a. **Cause.** Although the exact cause of the disease is unknown, it is accompanied by an increase in the numbers of two organisms--the fusiform bacillus and medium-size spirochetes (Vincent's spirillum). It is doubtful that NUG is readily communicable. Instruction in oral hygiene is not only an important preventive measure, but also an essential phase of the treatment. The infecting organisms can successfully invade and grow only in tissues whose resistance has been lowered. Therefore, proper diet, rest, exercise, and adequate oral hygiene can be good preventive measures.

b. **Effect.** Necrotizing ulcerative gingivitis is characterized by fetid breath and ulcerations covered by a whitish, yellowish, or gray pseudomembrane. They may be found in only a few areas or throughout the mouth. The most common site of these ulcers is the interproximal gingiva. The thin gray membrane covering the ulcer may be wiped off easily, exposing a highly inflamed area that bleeds very easily. There is a rapid destruction of the marginal and interproximal soft tissue. These tissues become so painful that it becomes difficult to brush the teeth and to masticate food. The onset of this infection is sudden, often with systemic symptoms of illness. In severe cases, there may be fever, an increased pulse rate, pallor of the skin, insomnia, and mental depression. Treatment includes procedures performed by the dental officer and home-care procedures performed by the patient.

1-40. PERICORONITIS

Pericoronitis is an inflammatory process occurring in gingival tissue found around the coronal (crown) portion of the teeth, particularly around partially erupted teeth. Similarly, operculitis is an inflammation of the gingival tissue flaps (operculi) found over partially erupted teeth. The most frequent site is the mandibular third molar region. The heavy flap of gingival tissues covering portions of the tooth crown of the tooth makes an ideal pocket for debris accumulation and bacterial incubation. In the acute phase, pain and swelling in the area are prominent features. Symptoms of a sore throat and difficulty in swallowing may be present. A partial contraction of muscles of mastication causing difficulty in opening the mouth (trismus) may also be experienced. Abscess formation in the area may occur, leading to marked systemic symptoms of general malaise and fever. Treatment should be directed toward careful cleansing of the pocket area and followup care with warm saline irrigations. Antibiotic therapy may be indicated if the condition warrants. The prognosis for retention of the tooth is dependent upon the possibility of complete elimination of the inflammation and elimination of the gingival flap.

Section VII. ABNORMALITIES OF ORAL MUCOSA

1-41. GENERAL

Oral mucosa has the same susceptibility to pathological change as does other covering tissue. Common abnormalities of the skin and the gastrointestinal tract may evidence themselves on oral mucosa. Local, focal oral mucosal lesions, generalized mucosal involvement, or intraoral lesions associated with a systemic problem may be caused by bacterial, fungal, or viral organisms. Benign or malignant lesions must always be considered when examining a patient's mouth.

1-42. VESICLE

A vesicle is a circumscribed, superficial elevation on the skin or mucous membrane containing fluid (serum, plasma, or blood). If the vesicle opens, it becomes an ulcer (an inflammatory lesion).

1-43. ULCER

An ulcer (figure 1-7) is an open sore of a superficial nature extending below the covering epithelial surface. The base of an ulcer is composed of granulation tissue resulting from initial healing. A secondary infection may develop in an ulcer, resulting in delay of the healing and repair process. A common cause of oral ulceration is trauma, which might even be a result of toothbrush injury. Irritation from a rough or broken tooth surface can also result in ulceration. Some ulcers start with vesicle formation.

NOTE: This painful ulceration on the lateral border of the tongue represents a nonspecific response to tissue injury. The cause of an ulcer must be determined and appropriate treatment initiated. Normal healing will often result without use of medication.

Figure 1-7. Ulcer.

1-44. PRIMARY HERPETIC GINGIVOSTOMATITIS

Initial exposure to the herpes simplex virus results in a generalized oral inflammation followed by vesicle formation and subsequent ulceration. Systemic symptoms of generalized illness accompany this initial attack. Most individuals have their primary exposure to this virus as infants; however, this disease may also occur in young adults and elderly patients. This condition is contagious. Healing occurs spontaneously with the virus remaining in the nerve tissue, lying dormant in a latent form. Future recurrence of the condition may be either intraoral or extraoral.

1-45. RECURRENT HERPES SIMPLEX: SECONDARY HERPETIC LESIONS

The herpes simplex virus may be reactivated (recurrent) in an extraoral form on the lips or, inside the mouth, in an intraoral form.

a. **Extraoral Herpes**. Cold sore blisters, also called herpes labialis (figure 1-8), are often associated with colds, trauma, fatigue, fevers, and prolonged exposure to the sun and the wind. The common site of occurrence is on the lips at the border with the skin of the face (called the vermilion border). The lesions usually consist of clusters of small vesicles that, in the early stage, contain a clear, transparent fluid. After a few hours, the vesicles rupture and form a crust or scab. The disease is self-limiting and usually disappears in 10 to 14 days. The individual is infective until complete healing has occurred.

NOTE: Cold sore blisters on the upper lip of this patient are caused by the herpes simplex virus. This recurring infection may be activated by prolonged exposure to sunlight and/or wind.

Figure 1-8. Extraoral herpes.

b. **Intraoral Herpes**. Intraoral herpes (figure 1-9) forms on extremely firm oral tissue surfaces such as the palate (roof of the mouth) and attached gingiva. Vesicles are not usually identified because they break down almost immediately into ulcers and coalesce to form multiple jagged ulcerations.

NOTE: The herpes simplex virus may cause ulcers to form inside the mouth. In this example, the ulcers are on the roof of the mouth (the palate).

Figure 1-9. Intraoral herpes.

c. **Complications.** The lesions may persist and be very serious in patients with a compromised immune system. While the virus may regress, it does not disappear. The lesions caused by the virus do disappear, however. Also, it is important for dental specialists to recognize that this may be a very serious infection when it occurs in immuno-compromised patients (AIDS, renal transplant, cancer chemotherapy, and so forth).

1-46. RECURRENT APHTHOUS ULCERS

Recurrent aphthous ulcer (RAU) (figure 1-10) is a chronic inflammatory disease with repeated episodes of ulcerations. Recent investigations seem to indicate that the aphthous lesion is associated with an altered local immune response. This disease is characterized by small, whitish ulcers with red borders. The disease normally occurs as a single lesion or, infrequently, as multiple lesions on the wet mucous membranes of the lip, tongue, cheek, or floor of the mouth. Lesions appear as depressions on the mucous membrane and are covered by a grayish-white or light-yellow membrane. There is no vesicle formation before the ulcer appears, distinguishing this disease from viral diseases of the oral mucosa. Associated with the development of a recurrent aphthous ulcer is generally trauma, endocrine change, psychic factors, or allergy. The lesions are painful; however, the condition is self-limiting with the lesions usually healing in 10 to 14 days without leaving scars. Recurrent aphthous ulcerative lesions are similar to the ulcerative herpes simplex lesions except that the herpes lesions can also occur on the attached gingiva.

NOTE: The formation of ulcers on movable mucosal surfaces is extremely painful to the affected individual. This condition can cause difficulty in eating. The patient must maintain normal nutrition and oral health habits to limit bacteria formation.

Figure 1-10. Recurrent aphthous ulcers.

1-47. ERYTHEMA MULTIFORME

Erythema multiform (figure 1-11) is an acute inflammatory condition that is easily observed because of a redness of the mucosa or the skin. It occurs in many forms on various parts of the body. Young adults are most commonly affected. The oral mucous membranes are frequently involved, including vesicle rupture that leaves painful oral ulcerations. The lips often exhibit crusted ulcerative lesions. Lesions appear rapidly (within 10 to 14 days) and persist several days or longer. The symptoms are treated, with spontaneous remissions occurring. Recurrence is common. If areas other than the skin and the mucous membranes are involved (such as the eye or the genitalia), the possibility of a syndrome complex exists.

NOTE: Raw, easily bleeding gingival tissues may represent an allergic response. In this patient, a pseudomembrane has developed over the gingiva. Lack of normal oral function and decreased oral cleansing caused by extreme discomfort allowed this buildup of ulcerative tissue.

Figure 1-11. Erythema multiform.

1-48. ALLERGIC MANIFESTATIONS

a. **Common Symptoms and Causes.** The allergic condition called angioneurotic edema may be related to food allergy, hypersensitivity, local infection, and endocrine or emotional disturbances. The characteristic symptom is rapid swelling of the affected tissue in 5 to 30 minutes with itching and burning sensations present. The areas most commonly involved are the skin about the eyes and chin, the lips, and the tongue. A major concern is the potential for laryngeal edema and airway compromise. The symptoms are treated.

b. **Adverse Reaction to Drug Absorption.** Ingestion of certain drugs by individuals having an idiosyncrasy or intolerance to them may result in the allergic manifestations referred to as stomatitis medicamentosa. The signs in the mouth vary from a sensitive erythema (redness) to an ulcerative stomatitis or gingivitis. If withdrawal of the suspected drug is followed by disappearance of the lesions, it is evidence of its causal relationships. Therefore, treatment consists of identification and elimination of the drug causing the lesions.

c. **Adverse Reaction to Contact With Drugs.** While the lesions of stomatitis medicamentosa are the result of absorption of drugs, the lesions of stomatitis venenata are caused by the direct contact with a drug or a material. Causative agents of stomatitis venenata may be topical medications, dentifrices, or mouthwashes. Intraoral signs may vary from a sensitive erythema (redness) to an ulceration. Treatment consists of elimination of the causative agent, local symptomatic care, or use of a mild antibacterial agent to minimize secondary infection of an ulceration.

1-49. LICHEN PLANUS

Lichen planus (figure 1-12) is a common inflammatory disease that is observed frequently on oral mucosa. Oral lesions appear on adults as a lacy network of slender white lines, primarily on the buccal mucosa. Initiating factors may be herpes simplex, various bacterial and fungal agents, drugs (penicillin and barbiturates), vaccinations, or radiation and chemotherapy. On the skin, lichen planus is observed as a bilateral symmetrical area on the flexor muscle surfaces of the wrists and lower leg. Lichen planus may exist in various oral forms--erosive, vesicular, or hypertrophic (increase in growth through cell size, not number). The symptoms are treated.

NOTE: The oral buccal mucosa is a common site for lichen planus. The picture shows the erosive form of lichen planus in which can be seen the typical lacy network of slender white lines on the mucosal surface.

Figure 1-12. Lichen planus.

1-50. LEUKOPLAKIA

Leukoplakia (figure 1-13) is a clinically descriptive term defining a white plaque. The white plaque is observed as irregular, thickening of the outer layers of the mucosa, a dull-white-to-gray color, variable size, and with very little pain unless ulceration and secondary infection have developed. The predominate oral cause is commonly associated with chronic irritation or trauma, such as wearing of ill-fitting dentures, cheekbiting, oral use of tobacco, or malpositioned or rough tooth surfaces. These areas are generally discovered by the dentist on routine examination, the patient being unaware of their existence. The condition may precede development of a malignant tumor. For this reason, early diagnosis and treatment is important. Leukoplakia resembles other conditions that may affect the oral mucosa; therefore, diagnosis must be based upon microscopic examination of cellular changes within the involved tissues. Tissue specimens are prepared for examination by a procedure called biopsy. Once a definitive diagnosis has been made, the treatment of choice may be removal of the lesion.

NOTE: This white plaque has formed in an area commonly associated with denture wear. Any area of leukoplakia that does not return to normal within 10 to 14 days after removal of the cause of irritation must be biopsied to confirm a benign cellular change.

Figure 1-13. Leukoplakia.

1-51. MONILIASIS OR CANDIDIASIS

Moniliasis of the oral mucosa membranes, also called candidiasis or thrush, is a surface infection resulting from a yeast-like fungus, *Candida albicans*. The lesion (figure 1-14) appears as deposits of pearly-white, roughened-surface plaque, which leaves a raw, red, painful surface when scraped off. Its treatment involves prescribing antifungal drugs. When natural resistance is lowered, this infection may appear and grow. Because it takes advantage of such conditions, moniliasis is known as an opportunistic infection. It may affect debilitated adult patients, infants, or patients receiving prolonged therapy with antibiotics or corticosteroids. In addition, moniliasis may be indicative of AIDS (acquired immunodeficiency syndrome). AIDS results from the human immunodeficiency virus (HIV).

NOTE: In this example, the yeast-like, white patches of the fungus *Candida albicans* can be clearly identified on the roof of the mouth.

Figure 1-14. Moniliasis.

1-52. DENTURE IRRITATION

Localized areas of inflammation associated with dentures can result from a number of factors. They may result from occlusal disharmonies, damage to the denture base or metal framework because of careless handling, or certain tissue changes. The mucous membrane beneath the denture becomes inflamed. If it is not remedied, this chronic irritation may result in hyperplasia of the oral mucosa. Hyperplasia (an increased growth of normal cells) caused by irritation at the border of the denture takes the form of long folds of excess tissue along the denture border. These folds are called epulis fissuratum. Irritation of the palate may result in the development of numerous papillae, a condition called inflammatory papillary hyperplasia. Treatment consists of reduction of the inflammatory component and surgical removal of any hyperplastic tissue. Prostheses correction is necessary to prevent recurrence.

1-53. ANGULAR CHEILITIS

Cheilitis is inflammation affecting the lips. Angular cheilitis usually begins as redness and peeling of the skin at the angles (corners) of the mouth. As the condition continues, cracks occur in the skin and mucous membranes at the commissure (corners) of the lips. This condition is usually caused by infection with *Candida albicans*. Other factors may also contribute to the lesions. These include vitamin B complex deficiency and decreased vertical dimension associated with inadequate dentures. Angular cheilitis is also a frequent finding in patients who have been infected with the human immunodeficiency virus (HIV).

1-54. AMALGAM TATTOO

Small, pigmented areas are common in the oral mucosa and are generally associated with tissue adjacent to restored teeth. During the placement of fillings, amalgam may find its way into soft tissue causing a discoloration. If clinical diagnosis can establish a definite diagnosis of amalgam tattoo, no treatment is necessary. An amalgam tattoo (figure 1-15) may be associated with the alveolar ridge or other mucosal tissue in patients without teeth. If there is a doubt concerning the diagnosis, a biopsy should be done. Pigmented soft tissue tumors are not frequently found on oral mucosa; though, when found, they range from a benign freckle to malignant melanoma.

NOTE: This pigmented palatal lesion is not directly associated with the dentition. Amalgam particles may be found throughout the oral cavity. Amalgam particles can generally be identified as such on radiographs. Any questionable cause of tissue discoloration should be evaluated histologically after biopsy.

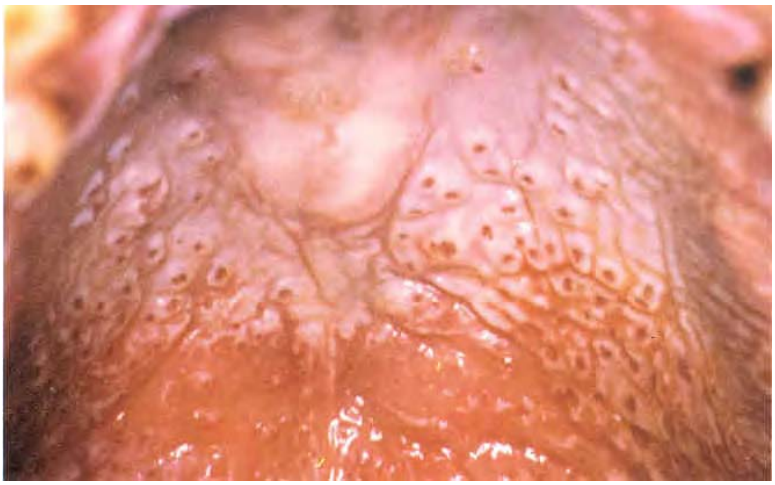
Figure 1-15. Amalgam tattoo.

1-55. ASPIRIN BURN

Aspirin burn is a chemical burn caused by holding aspirin tablets against the mucous membranes to relieve toothache. The treatment is usually palliative (medications are used to relieve discomfort, but not to cure the lesions) and the prevention of secondary infection is considered.

1-56. NICOTINIC STOMATITIS

Nicotinic stomatitis (figure 1-16) is caused by smoking, especially pipe-smoking. The irritation from heat and combustion products stimulates increased production of new epithelial cells resulting in hyperkeratosis (thickening of the layer of keratin on the epithelium) of the mucosal surface of the palate. The ductal openings of the minor salivary glands become inflamed, forming tiny spots, points, or depressions (a punctate appearance). Treatment requires elimination of the smoking habit.



NOTE: In this example, notice the spots. With elimination of the smoking habit, a return to normal might be expected. There is no documented malignant cellular change associated with this condition. However, there is likely to be some abnormality in tissue texture over a prolonged time.

Figure 1-16. Nicotinic stomatitis.

1-57. SNUFF DIPPER'S POUCH

Another oral lesion commonly seen in users of chewing tobacco or dip is called the snuff dipper's pouch. When the tobacco is kept in touch with oral mucosa over prolonged time, hyperkeratosis (thickening) occurs. Most tobacco dippers keep their chew in the mandibular labial vestibule, but the lesion can appear wherever the tobacco is kept. The lesion can be white-gray in color and develops deep folds of excess tissue. Treatment requires the elimination of the dipping habit. If the habit is not eliminated, the lesion can progress to cancer. A biopsy may be indicated to rule out cancer.

1-58. FORDYCE'S SPOTS

Fordyce's spots, or Fordyce's granules, are caused by the entrapment of normally functioning sebaceous glands during development of the embryo. These spots are generally seen in the buccal mucosa in the retromolar area. There is no abnormality of the thin epithelial covering. No inflammation is present. The granules are observed as small, rounded elevations (maculopapules) of a yellowish-white color. They may occur singly (isolated) or in clusters. There is no clinical significance associated with Fordyce's spots. The granule is not harmful and, generally, no treatment is indicated.

1-59. ORAL TORI

In about one-fifth of the population, benign bony outgrowths called tori (figure 1-17) occur in the midline of the palate or on the lingual surfaces of the mandible in the region of the cuspids and bicuspids. Tori are covered with relatively thin mucosa. The names torus palatinus and torus mandibularis indicate the location of these benign bulges of excess bone. Oral tori develop slowly and do not need to be removed unless they interfere with speech, denture design, or are repeatedly irritated during normal mastication.

Figure 1-17. Oral tori.

NOTE: Bilateral mandibular oral tori of varying sizes are frequently observed. These are within a normal variation of bone growth and no pathologic significance is associated with their presence.

Continue with Exercises

EXERCISES, LESSON 1

INSTRUCTIONS. The following exercises are to be answered by marking the lettered response that best answers the question or best completes the incomplete statement or by writing the answer in the space provided.

After you have completed all the exercises, turn to "Solutions to Exercises" at the end of the lesson and check your answers.

1. List the four major signs of inflammation.

   a. _____

   b. _____

   c. _____

   d. _____

2. Sometimes, in addition to the four signs above, there is a fifth sign of inflammation.

   This is a _____ of _____.

3. List four beneficial effects of inflammation.

   a. _____ _____ _____ the irritants present in the area.

   b. _____ _____ _____ and often _____ bacteria, dead cells, and other debris.

   c. _____ _____ toxic substances.

   d. Clotting of the edema fluid _____ _____ _____ _____ and prevents the inflammation from spreading.

4. When inflammation is not contained within a localized area, but spreads throughout a tissue, the condition is called:

   a. Suppuration.

   b. An abscess.

   c. Cellulitis.

   d. Hyperemia.

5. Dental caries is a specific disease which brings about the dissolution and disintegration of the_____ _____ of the tooth, that is, _____ , _____ , and _____.

6. From the following diseases that affect the pulp, select the one <u>least</u> commonly encountered in clinical dental practice.

   a. Acute pulpitis.

   b. Chronic pulpitis.

   c. Pulp abscess.

   d. Hyperplastic pulpitis.

7. Select the one item that is <u>NOT</u> a degenerative change of the pulp of the tooth.

   a. Fibrosis.

   b. Hyaline degeneration.

   c. Calcific degeneration.

   d. Pulp stones.

   e. Pulp abscess.

8. In caries of the dentin, more dentinal tubules are found in the:

   a. Infected layer.

   b. Affected layer.

9. Caries in enamel may be caused by:

   a. Acidogenic agents.

   b. Proteolytic bacteria.

   c. Lamella tract dissolution.

   d. "a," "b," and "c."

   e. "a" and "b."

SPECIAL INSTRUCTIONS FOR EXERCISES 10 THROUGH 15. Match the name of the condition or disease in Column B to its description in Column A and write your answer in the space provided at the beginning of the exercises..

COLUMN A	COLUMN B
___ 10. Cyst associated with root of a tooth	a. Hyperplastic pulpitis
___ 11. Loss of tooth structure through physiological or pathological means, which can be observed on a radiograph	b. Cronic apical perindontitis
___ 12. Mechanical wearing away of tooth structure	c. Radicular cyst
___ 13. A soft pulpable mass overlying the pulp chamber within the crown of the tooth	d. Erosion
___ 14. Chronic inflammation of the periapical tissue	e. Abrasion
___ 15. Chemical wearing away of tooth structure	f. Resorption

SPECIAL INSTRUCTIONS FOR EXERCISES 16 THROUGH 20. Match the name of the condition or disease in Column B to its description in Column A and write your answer in the space provided at the beginning of the exercises.

COLUMN A	COLUMN B
___ 16. Inflammation of gingival tissue	a. NUG or Vincent's disease
___ 17. Inflammation of the tissues supporting the teeth	b. Pericoronitis
___ 18. Inflammation of the gingiva around the crown of a partially erupted tooth	c. Periodontitis
___ 19. Abscess formed along the side of the tooth caused by irritation in the gingival sulcus	d. Gingivitis
___ 20. Bacterial infection, commonly called "trench mouth," characterized by fetid breath, ulcerative gingival tissue, and grayish pseudomembrane	e. Periodontal abscess

21. When the inflammation in the periapical tissues is very severe and results in swelling, fever, and sharp pain and the pathology can be seen in the periapical bone on the radiograph, the condition is:

    a. Acute apical abscess.

    b. Chronic apical periodontitis.

    c. Phoenix abscess.

    d. Acute apical periodontitis.

22. Extension of infection from carious teeth, salivary glands, or tonsils may cause a firm swelling of the floor of the mouth and elevation of the tongue. This condition is:

    a. Necrotizing ulcerative gingivitis.

    b. Acute apical abscess.

    c. Acute pulpitis.

    d. Periodontitis.

    e. Ludwig's angina.

23. Benign bony outgrowths in the mouth that are covered with relatively thin oral mucosa, are called:

    a. Fordyce's granules.

    b. Opportunistic infections.

    c. Oral tori.

    d. Dental granulomas.

    e. Osteomyelitis.

24. Is an aspirin burn an example of stomatitis venenata?

    a. Yes.

    b. No.

25. A dental specialist sees pearly-white, roughened-surfaced plaque in a patient's mouth. This lesion, caused by a yeast-like fungus, is:

    a. Leukoplakia.

    b. Trench mouth.

    c. Candidiasis.

    d. Intraoral herpes.

    e. Stomatitis aphthosa.

26. A dental specialist sees hyperkeratosis and a white-gray lesion in the mandibular labial vestibule. This lesion is:

    a. Angular cheilitis.

    b. Denture irritation.

    c. Erythema multiforme.

    d. Snuff dipper's pouch.

    e. Recurrent aphthous ulcers.

27. When cracks occur in the skin and mucous membranes at the commissure of the lips, this condition is:

    a. Herpes labialis.

    b. Angular cheilitis.

    c. Candidiasis.

    d. Cellulitis.

    e. Granulation.

SPECIAL INSTRUCTIONS FOR EXERCISES 28 THROUGH 32. Match the name of the condition or disease in Column B to its description in Column A and write your answer in the space provided at the beginning of the exercises.

COLUMN A	COLUMN B
___ 28. Dermatological disease whose oral manifestations appear as a lacy network of slender white lines, primarily in the buccal mucosa	a. Secondary herpetic lesions
___ 29. Small pigmented areas commonly found in the oral cavity and generally associated with the tissues adjacent to restored teeth	b. Recurrent aphthous ulcers
___ 30. Sebaceous glands that are found on the oral mucosa of the cheek and lip	c. Lichen planus
___ 31. Ulcerative lesion found on the intraoral Mucosa that occurs without vesicle formation	d. Amalgam tattoo
___ 32. Cold sore blisters and mouth ulcers caused by the herpes simplex virus	e. Fordyce's spots

Check Your Answers on Next Page

SOLUTIONS TO EXERCISES, LESSON 1

1. a. Swelling
   b. Pain
   c. Heat
   d. Redness (para 1-4)

2. disturbance; function (para 1-4)

3. a. Increased fluid dilutes
   b. Blood cells engulf; digest
   c. Antibodies neutralize
   d. walls off the area (para 1-5)

4. c (para 1-9)

5. hard structures; enamel; cementum; dentin (para 1-15)

6. d (para 1-25)

7. e (para 1-24)

8. b (para 1-18c)

9. e (para 1-16)

10. c (para 1-32c, figures 1-2, 1-3)

11. f (paras 1-14)

12. e (para 1-13)

13. a (para 1-25)

14. b (para 1-29)

15. d (para 1-12)

16. d (para 1-35)

17. c (para 1-36)

18. b (para 1-40)

19. e (para 1-38)

20. a (para 1-39)

21. c (paras 1-31, 1-30)

22. e (para 1-33)

23. c (para 1-59)

24. b (paras 1-48c, 1-55)

25. c (para 1-51)

26. d (para 1-57)

27. b (para 1-53)

28. c (para 1-49)

29. d (para 1-54)

30. e (para 1-58)

31. b (para 1-46)

32. a (para 1-45)

End of Lesson 1

LESSON ASSIGNMENT

LESSON 2	Diseases of the Soft Tissue.
LESSON ASSIGNMENT	Paragraphs 2-1 through 2-37.
LESSON OBJECTIVES	After completing this lesson, you should be able to:

2-1. Identify oral tumors and biopsy technique.

2-2. Identify abnormalities of the tongue.

2-3. Identify developmental anomalies of the teeth.

2-4. Identify the signs and symptoms of systemic diseases from what can be observed in the oral cavity.

2-5. Identify diseases that can be transmitted in the dental clinic.

SUGGESTION After studying the assignment, complete the exercises at the end of this lesson. These exercises will help you to achieve the lesson objectives.

LESSON 2

DISEASES OF THE SOFT TISSUE

Section I. ORAL TUMORS (NEOPLASMS) AND LABORATORY STUDIES

2-1. ORAL TUMORS

a. **Definition.** A neoplasm (tumor) is an abnormal growth of new tissue that grows independently of surrounding structures and serves no physiological purpose. A tumor can usually be removed surgically. Since benign tumors grow by expansion, when removed, they are less likely to recur than malignant tumors. Malignant tumors (cancers) grow by extension into the surrounding contiguous tissues. They tend to spread (metastasize) to other parts of the body by way of the blood vessels, lymphatic system, or membranous surfaces.

b. **Examining the Oral Cavity.** The oral cavity is a common site for many types of tumors. Early treatment of tumors is essential. Examination and diagnosis by the dental officer must include a consideration of suspicious lesions, such as leukoplakia, and conditions that might develop into tumors. Any abnormal condition seen by a dental specialist, such as unexplained swelling, abnormal discoloration, or ulceration, should be reported to the dental officer immediately.

2-2. FIBROMA

Fibromas (figure 2-1) are the most common benign tumor growths in the oral cavity. They are a connective tissue response to irritations resulting in a well-defined, slow-growing firm mass. The overlying tissue (oral mucosa) is usually normal in appearance and color. A common site for irritation fibromas is the buccal mucosa, but they may be found throughout the oral cavity. After surgical removal, there is no recurrence.

NOTE: The biting line of the buccal mucosa is a common area for fibroma development. Fibromas are benign, mucosal-covered, traumatically-stimulated growths of fibrous connective tissue which are removed surgically by excision.

Figure 2-1. Fibroma.

2-3. PAPILLOMA

Papillomas (figures 2-2 and 2-3) are benign stalk-like (pedunculated) growths originating from the surface tissues. This outward growing, cauliflower-like, surface tumor is common within the oral cavity and may occur anywhere on oral mucosa. Surgical removal is curative.

NOTE: The lingual frenum of the tongue is a common site of papilloma development. This possibly is because of constant irritation of this tissue by irregular or sharp lower incisor teeth. Surgical removal is indicated. Notice the cauliflower shape of the papilloma.

Figure 2-2. Papilloma, cauliflower shape.

NOTE: This papilloma does not have the normal warty (cauliflower-like) appearance. The biopsy confirmed an epithelial tissue abnormality. Variation in appearance from the classically-described lesion should always be considered in evaluation of tissue irregularity. Multiple papillomas may indicate secondary syphilis.

Figure 2-3. Papilloma, variation in appearance.

2-4. PYOGENIC GRANULOMA

A pyogenic granuloma (figure 2-4) may develop as a part of the wound repair process that has been modified by the hormonal changes of puberty or pregnancy. Pyogenic granulomas appear as hemorrhagic tissue growths (bright red in appearance) and are usually found on gingival tissue in pregnant females in response to irritation from subgingival calculus. Surgical removal of the tumor mass and removal of the causative factor result in normal healing. However, if the irritant is not removed, the pyogenic granuloma may recur.

NOTE: The high degree of vascularity can be seen in the bright red appearance of this pyogenic granuloma. Ease of bleeding is characteristic. Irritation can be a cause. Removal of the lesion is necessary, but the causative factor must also be identified and removed.

Figure 2-4. Pyogenic granuloma.

2-5. ODONTOMA

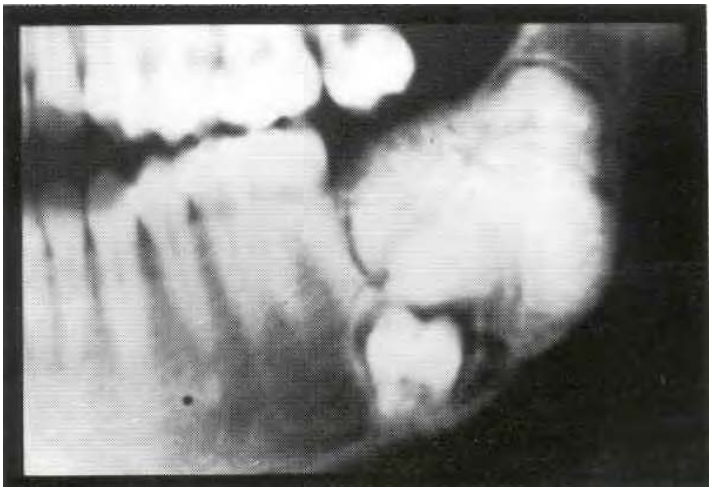
An odontoma is a benign odontogenic tumor of mixed tissue origin that is located within the facial bones. An odontoma is usually diagnosed from a radiograph. Odontomas appear as either irregular masses of calcified material or as numerous tooth-like structures.

a. **Compound Odontomas**. Compound odontomas (figure 2-5) have a similarity to normal teeth and are generally found in the anterior arch (canine and incisor teeth).

b. **Complex Odontomas**. Complex odontomas (figure 2-6) are irregular masses and are generally found in the posterior dental arch (premolar and molar teeth).

NOTE: This compound odontoma has resulted in the lack of eruption of the permanent central incisor and retention of the deciduous central incisor. A recognizable tooth form with abnormal crown enamel development and enamel pearl can be observed.

Figure 2-5. Compound odontoma.



NOTE: This large complex odontoma consists of dental structures (dentin, cementum, enamel, pulpal tissues) and has no recognizable tooth form. Note the displacement of the permanent second molar and lack of third molar development.

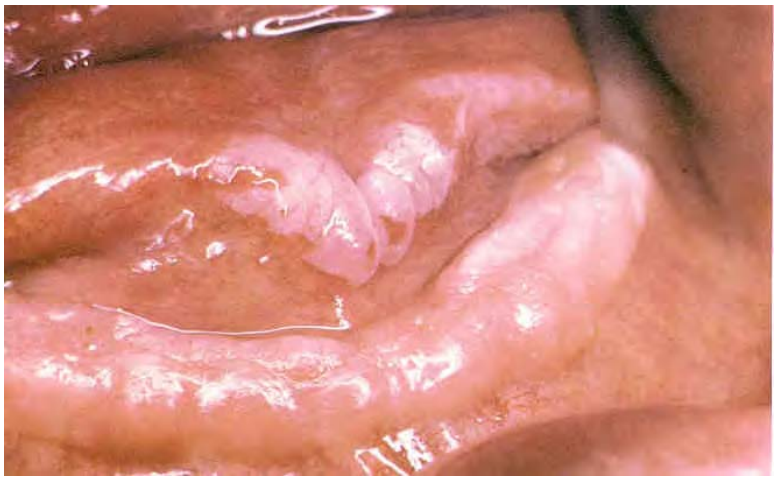
Figure 2-6. Complex odontoma.

2-6. ORAL CANCER

The most common form of oral cancer is squamous cell carcinoma. It is most commonly found on the lips, especially the lower lip. Inside the mouth, the posterior lateral border of the tongue is the most common site for squamous cell carcinoma, followed by the floor of the mouth. Oral cancer (figures 2-7 and 2-8) may appear as leukoplakia (white plaque), erythroplakia (velvet-red plaque), or an ulceration. The red patches (plaques) have a much higher potential of becoming malignant than do the white plaques. Oral cancer is painless during the initial phase of development. It is generally found in older individuals, occurring with greater frequency in males. Metastatic potential varies with location. Cancer of the lip tends to metastasize later in development, while cancer of the tongue tends to metastasize early. Tobacco is regarded as the most important contributing factor for developing oral cancer. With the use of smokeless tobacco, there is a significantly higher risk of developing oral cancer. Since alcohol consumption also appears to add to the risk of oral cancer development, lifestyle habits that mix smoking and drinking impose even greater risks for developing oral cancer.

NOTE: The lower lip is a common site of oral cancer. Lesions are often present for an extended time before being evaluated. Often, a small chronic scab may be the only abnormality. Most affected lips show evidence of continuous solar and ultraviolet light damage to the tissues.

Figure 2-7. Oral cancer, lower lip.



NOTE: The clinical appearance of leukoplakia revealed a squamous cell carcinoma on histologic examination. The mouth floor is a common site for oral cancer. All oral tissues must be evaluated for abnormality with each oral examination so that malignant growths may be recognized early in development.

Figure 2-8. Oral cancer, floor of the mouth.

2-7. BIOPSY

a. **General.** Biopsy is the procedure for obtaining a tissue specimen for microscopic examination in order to establish a diagnosis. This procedure is used when tissue growth cannot be diagnosed by clinical observation alone. Biopsy is extremely important in diagnosing malignant growths (tumors) requiring early treatment and in identifying other conditions requiring early treatment or specific types of treatment. Surgical excision (complete removal of involved tissues) or removal of a part of the tissue may be done. Excision is the method of choice when the lesion is small and complete removal is the treatment.

b. **Processing the Specimen.** After the tissue specimen has been removed surgically, it should be placed in a container with 10 percent formalin. The container should be large enough to accommodate fixative solution equal to about 20 times the volume of the specimen. Usually 48 to 72 hours is enough time to ensure adequate fixation. However, the time will be determined by the size of the specimen. The specimen, a properly completed Standard Form 515 (Medical Record-Tissue Examination) with five copies, a clinical history, and appropriate dental radiograph should all be forwarded to the oral-maxillofacial pathologist.

NOTE**:** The container must be properly identified in case it is separated from the SF 515, to include the patient's name and social security number, type of tissue sample (for example, soft tissue, bone), doctor's name, clinic address, and date.

Section II. ABNORMALITIES OF THE TONGUE

2-8. GENERAL

The tongue may be affected by most oral soft tissue lesions, but certain lesions are peculiar to the tongue. These lesions may be the result of developmental anomalies, systemic disorders, local irritations, or neoplastic changes. Because of the tendency for oral cancer to occur in the tongue, a thorough examination must be made to ensure early discovery of these lesions.

2-9. MEDIAN RHOMBOID GLOSSITIS

Median rhomboid glossitis (figure 2-9) appears as a smooth, flat, depressed or elevated nodular area on the dorsum of the tongue just anterior to the circumvallate papillae. It is usually an oval or diamond-shaped area and stands out because the area has no filiform papilla. Median rhomboid glossitis is believed to be caused by a *Candida* infection, often with secondary hyperplasia. Treatment may include the use of an antifungal drug and surgical removal of the hyperplastic tissue.

Figure 2-9. Median rhomboid glossitis.

2-10. DEVELOPMENTAL ANOMALIES

a. **Cleft or Bifid Tongue.** This condition is characterized by the failure of the two halves of the tongue to unite. It rarely occurs. The cleft tongue is usually normal in size, but its function is greatly impaired. Treatment is surgical, if indicated.

b. **Ankyloglossia**. In ankyloglossia (figure 2-10), the tongue is restricted in its movements by a strand of mucosa (lingual frenum) that attaches the anterior third of the tongue to the floor of the mouth and the lingual gingival mucosa. Persons with this condition are commonly called "tongue-tied." Treatment is surgical.

Figure 2-10. Ankyloglossia.

c. **Geographic Tongue**. Geographic tongue, or benign migratory glossitis, is characterized by alternating red areas with a yellowish-white border (figures 2-11 and 2-12. This appearance is caused by alternating areas of hypertrophy and atrophy of the filiform papillae. In the areas of atrophy, the fungiform papillae appear as irregular, reddish areas surrounded by horny growth (keratosis). In the areas of hypertrophy, filiform papillae appear as whitish areas. The patterns developed are variable with changes in shape and position from time to time. The cause of this lesion is unknown. Developmental defects may also be present. These defects are responsible for a secondary burning sensation caused by debris collection, Treatment consists of proper cleansing of the tongue.

Figure 2-11. Geographic tongue, dorsal view.

Figure 2-12. Geographic tongue, lateral view.

d. **Fissured Tongue.** In fissured (or scrotal) tongue, the surface of the tongue appears furrowed with a deep median fissure and numerous shorter fissures radiating out on either side or it may be seen with independent furrows (figure 2-13). This condition is usually painless. However, with food accumulation, pain may result. Proper cleansing of the tongue is essential.

Figure 2-13. Fissured tongue.

e. **Macroglossia.** A congenital macroglossia is generally caused by an overdevelopment of the muscular portion of the tongue. Surgical correction is the treatment indicated for severe cases. Macroglossia may develop after removal of teeth. This develops as a hypertrophy (increase in cell size) when the teeth no longer contain the tongue within the previously established boundaries.

f. **Aglossia.** In this condition, a portion or all of the tongue is absent. Rarely is all the tongue absent.

2-11. HAIRY TONGUE

Hairy tongue (or black hairy tongue) occurs on the dorsum of the tongue. The filiform papillae are hypertrophied and may be colored by substances in the diet. Treatment consists of good oral hygiene and brushing the tongue.

2-12. SYSTEMIC SMOOTH TONGUE

Systemic disorders may cause a smoothness of the tongue because of atrophy of papillae on the dorsal surface. Associated color change may denote the possible underlying cause. A bright, "beefy-red" tongue is associated with pernicious anemia, pellagra, or nicotinic acid (niacin) deficiency. Deficiencies of vitamin B and niacin may cause a magenta-colored atrophic tongue. Because of the papillae atrophy, these patients generally complain of a burning sensation as a symptom of this disorder. Patients with burning, smooth tongues should be evaluated for vitamin B_{12} deficiency or other systemic causative factors.

Section III. DEVELOPMENTAL ANOMALIES OF THE TEETH

2-13. GENERAL

Developmental anomalies of teeth are marked deviations from the normal standards in color, contour, size, number, and degree of development of the teeth. Systemic as well as local factors may operate to produce these developmental disturbances. Such influences may begin before or after birth so that either deciduous or permanent teeth may be involved. Usually, it is the permanent teeth that are influenced and, in all instances, only those not completely formed at the time of the disturbance.

2-14. HYPOPLASIA

In hypoplasia (figure 2-14), there is a lack of development or a defective development of the enamel of the tooth before its eruption. In deciduous teeth, enamel hypoplasia can be caused by a disturbance in the enamel formation before birth and, for some deciduous teeth, after birth. In permanent teeth, enamel hypoplasia can only be caused by some disturbance after birth since enamel formation of the permanent dentition begins at birth. Enamel prisms are deposited by the enamel organ in a definite pattern to form the crown of the tooth. A local disturbance may interfere with this process and result in defective development. The degree of the defect (hypoplasia) varies from mild, shallow depressions or grooves to extensive grooves or pits arranged in horizontal rows around the crown. These grooves or pits extend into the enamel as far as the dentinoenamel junction. The defect may be a lack of development of all or part of the enamel, leaving exposed dentin. Hypoplastic enamel and poorly formed dentin can be attributed to various causes or combinations of causes, such as infectious diseases (scarlet fever, measles, pneumonia), rickets, or hereditary factors. Trauma to primary teeth may cause a localized hypoplastic defect in a developing permanent tooth (called Turner's tooth). Prevention is much better than treatment. A physician who provides good health supervision (including proper treatment of infant disease) and good nutrition are the keys to prevention.

NOTE: Interruption of the enamel developmental process results in irregular enamel formation or lack of enamel formation. Restorative treatment may be required because of susceptibility to decay and to improve appearance.

Figure 2-14. Enamel hypoplasia.

2-15. ANODONTIA

Anodontia, which means absence of teeth, is an anomaly in which the enamel organ fails to form. Some (partial anodontia) or all (total anodontia) of the teeth may fail to develop. Cases in which only part of the tooth fails to form are considered forms of hypoplasia.

2-16. ENAMEL HYPOCALCIFICATION

Defective development of the enamel matrix may cause hypocalcification. This implies a poor quality of enamel, although the amount is normal. The enamel may be chalky, crumble easily, and contain white or brown areas or spots. Dental fluorosis is a form of hypocalcification.

2-17. MOTTLED ENAMEL

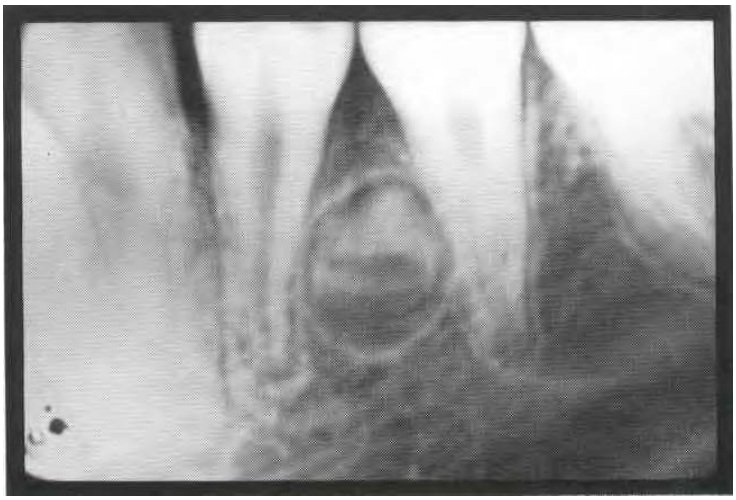
Mottled enamel (figure 2-15), also called dental fluorosis, is a form of enamel hypocalcification which results from the ingestion of excessive fluoride during the period of enamel formation. To cause mottling, fluoride must be present in concentrations several times that found in controlled fluoridated water supplies. The mottling of enamel varies in extent and severity, depending on the amount of fluoride in the water and how long it was routinely used. In mild cases, the enamel is opaque but of good structure. In severe cases, it may be chalky and crumble easily. The area of involvement varies from spots on a few teeth to extensive mottling of many teeth. The mottled areas may have a white, opaque appearance or they may have a stained appearance ranging from yellowish-brown to dark-brown. This condition is significant because it makes teeth unsightly. Superficial bleaching and acid etch composite may be used to correct the condition. Severe cases may require porcelain veneers or crowns.

NOTE: When teeth are discolored by fluoride, treatment may be required to improve appearance.

Figure 2-15. Mottled enamel.

2-18. SUPERNUMERARY TEETH

Dental hyperplasia may result in additional enamel organs being formed, resulting in development of teeth beyond the normal number (32) permanent teeth. Hyperplasia is defined as an increase in the number of individual tissue elements. This may also account for supernumerary cusps or roots within the normal complement of teeth (see figure 2-16). The vast majority of supernumerary teeth occur in the maxilla, with the most common being a mesiodens (a tooth located between the permanent maxillary central incisors) (see figure 2-17).



NOTE: This radiograph reveals a developing supernumerary (third) premolar.

Figure 2-16. Supernumerary teeth, premolar.

NOTE: A mesiodens is in evidence between the two normal central incisors. Note its conical shape. Malposition of teeth and generalized gingivitis are other features of this example.

Figure 2-17. Supernumerary teeth, mesiodens.

2-19. OTHER DEVELOPMENTAL ANOMALIES

a. **Dens in Dente.** In this condition, during the developmental state, a tooth within the original tooth is formed. The new tooth is usually composed of enamel and sometimes includes dentin and cementum.

b. **Concrescence.** In this condition, after the roots are developed, cementum is deposited between two adjoining teeth and joins them together. Only the roots are united. The crowns are always separate.

c. **Fusion.** In this condition, two developing and adjoining teeth join to form a single large crown. Fusion may occur between two normal adjoining teeth or between a normal tooth and a supernumerary tooth. Fusion may involve the crown and root (total fusion) or the roots may be separate. Usually, there are two distinct pulp chambers. In fusion of the root, the dentin of the two teeth is continuous as opposed to concrescence.

d. **Gemination.** In gemination, or twin formation, a double crown is attached to a single root. The crown appears twice as wide as normal with a shallow groove through the center.

e. **Enamel Pearls (Enamel Drops).** In this condition, small islands of enamel, 1 to 2 mm in diameter, are located on the root surface close to the cementoenamel junction. If covered by cementum, they probably will not be noticed on the extracted tooth except histologically. They are of significance only as potential problems in development of periodontal lesions.

Section IV. ORAL MANIFESTATIONS OF SYSTEMIC DISEASES

2-20. GENERAL

Many systemic diseases may develop manifestations in the oral cavity, sometimes before it is evident in any other part of the body.

2-21. MEASLES

This is an acute, contagious disease caused by a virus. It is transmitted by saliva droplets via the respiratory tract. The disease usually occurs in children and the initial episode provides immunity to further attacks of the disease. Koplik's spots appear several days before the characteristic skin lesions of measles. The spots are bluish-white and surrounded by an inflamed red zone. These spots are formed on the buccal mucosa opposite the molars.

2-22. MUMPS

This condition is an acute, contagious viral infection. It is characterized chiefly by swelling of the salivary glands, usually the parotid gland. Symptoms of the disease include fever, chills, headache, and painful swelling below the ear. Salivation and opening the mouth cause discomfort. The parotid duct opening in the buccal mucosa is usually reddened and swollen. The saliva itself contains the virus.

2-23. SCURVY

This is a rare condition caused by a deficiency of vitamin C in the diet. Scurvy is marked by anemia, spongy gums, and a tendency for bleeding by the mucous tissues. The gingiva may have a purplish color.

2-24. PERNICIOUS ANEMIA

This condition is a result of a vitamin B_{12} deficiency in the diet. Symptoms include generalized weakness, numbness or tingling of the extremities, and a sore, painful tongue. The tongue is generally inflamed and often described as beefy-red in color. Treatment consists of the ingestion of vitamin B_{12} and folic acid.

2-25. EPILEPSY

The disease epilepsy is discussed here because of the use of Dilantin in its treatment and the marked gingival hyperplasia that may result (figure 2-18). Hyperplasia is rapid growth of the dense, fibrous connective tissue of the gingiva. In advanced hyperplasia, the teeth may be completely covered by firm, light pink gingival tissue. Normally, bleeding, inflammation, or secondary infection is not a problem in these cases. Treatment of the hyperplastic tissue is surgical removal and strict oral hygiene care to prevent recurrence.

NOTE: Gingival tissue overgrowth of the clinical crown is caused by Dilantin therapy in some patients. Poor oral hygiene contributes to this development, with the fibrous tissue compounding the hygiene problem.

Figure 2-18. Dilantin hyperplasia.

2-26. CANDIDIASIS (MONILIASIS; THRUSH)

The causative agent of candidiasis is a yeast-like fungus called *Candida albicans*. This condition is characterized by lesions that appear on the oral mucosa as white or gray-white patches resembling curdled milk. Limited numbers of this fungus normally occur in the mouths of healthy persons but can cause disease in newborn infants, patients receiving antibiotics, and patients who are debilitated because of alcoholism, leukemia, diabetes, or acquired immunodeficiency syndrome (AIDS).

2-27. LEUKEMIAS

Leukemias are a group of often-fatal diseases that may show early oral symptoms. They are characterized by marked increases in the number of leukocytes, many of which do not mature. Leukemias may vary from acute to chronic, with the acute form being more common in young persons and the chronic form in older persons. Acute leukemia in a young person responds very well to chemotherapy and five-year cure rates are the rule. Oral symptoms of leukemias include hemorrhaging; ulceration; and enlargement, spongy texture, and magenta coloration of the gingiva. Enlargement of lymph nodes, symptoms of anemia, and hemorrhagic tendencies are also typical. The diseases occur in myelogenous, lymphatic, and monocytic forms. These forms are for the type of white blood cell that is most predominant in the disease.

2-28. DIABETES MELLITUS

Diabetes mellitus is a disease in which the body is unable to use carbohydrates (primarily sugar) caused by a deficiency in the insulin secretion of the pancreas. This disease is controlled by strict dietary restriction supplemented, as needed, by insulin or other drugs. This metabolic disorder lowers tissue resistance to infection and retards healing. Diabetics also appear to be more susceptible to periodontal disease. This disease may be accompanied by a tendency of gingival tissues to bleed easily.

2-29. ADDISON'S DISEASE

This disease is characterized by increased deposits of melanin in the tissue. Pigmentation of the lips, mucosa, and gingiva are common. Addison's disease is caused by insufficient secretion of the adrenal glands. Since these patients are very sensitive to minor stresses, dental procedures must be carried out with proper precautions.

2-30. HEMOPHILIA

Hemophilia is a condition in which bleeding is profuse because of inadequate clotting factors. The lack of the clotting mechanism results in prolonged, uncontrolled bleeding. This condition is a sex-linked hereditary disease confined mainly to males, but it is transmitted by females. The eruption of teeth or extraction or exfoliation of teeth may be accompanied by bleeding, possibly lasting days. Since death could result from hemophilia, recognition of it is important before even minor dental procedures are attempted. These patients should be treated under controlled conditions, such as at a hospital dental clinic.

Section V. TRANSMITTABLE DISEASES

2-31. GENERAL

The dental specialist must be aware of some diseases that can be transmitted in the dental clinic. Being aware of these diseases will make sterilization and disinfection procedures more meaningful.

2-32. INFLUENZA AND THE COMMON COLD

Influenza, the common flu, is spread by airborne droplet viruses that consist of many different antigenic types. Therefore, it is difficult for a vaccine to be completely effective. New antigenic types are produced by mutation of the viruses. The common cold is caused by many different viruses (possibly hundreds), each of which may have antigenic types.

2-33. HEPATITIS

Hepatitis is a disease caused by a virus that damages the liver. In severe cases, liver necrosis (death of tissue) is common. The two types of distinguishable hepatitis are infectious hepatitis (Type A) and serum hepatitis (Type B). Infectious hepatitis is usually spread by fecal contamination of food or drink. It has an incubation period of about 30 days. Serum hepatitis can be spread by punctures of the tissue with needles and dental scalers that have come in contact with contaminated blood or serum. It has a much longer incubation period (perhaps several months) than infectious hepatitis. The risk of infection is greater between patients than between patient and dental specialist. All dental specialists should be tested and immunized against Type B hepatitis if they are not already immune.

2-34. TUBERCULOSIS

Tuberculosis is caused by a nonsporeforming bacillus bacteria, *Mycobacterium tuberculosis*. Cross-infection most likely occurs by droplet transmission. Active tuberculosis infects the individual with a virulent (a strong disease-causing organism) strain of bacteria that the patient cannot ward off by natural means. Inactive tuberculosis occurs when the bacteria are walled off by the fibrous tissue. Even though the Tine and the Mantoux tests detect the presence of circulating antibodies, these tests do not tell whether the infection is active or inactive. A positive test result merely indicates either a present or previous infection with the tuberculous bacteria. A positive test result should always be reported to the dental or medical officer. Tuberculosis of the oral mucosa occurs if wounds or erosions become infected by tubercle bacilli contained in the sputum. The oral lesions vary greatly in their appearance. The tongue is the most frequent site for these lesions. Antibiotics are effective in the treatment of certain types of tuberculosis. However, multi-drug resistant strains have been discovered recently. Outbreaks of tuberculosis have occurred in areas with a high HIV population.

2-35. ACQUIRED IMMUNODEFICIENCY SYNDROME

Acquired immunodeficiency syndrome (AIDS) is a severe condition caused by infection with the human immunodeficiency virus (HIV). Infected patients may have a variety of manifestations ranging from no symptoms at all to severe immunodeficiency and life-threatening secondary infectious diseases. There are three stages to this disease--asymptomatic infection HIV, AIDS-related complex (ARC), and AIDS. The serum test for the HIV antibody is not necessarily "positive" in all three stages of HIV infection. In stage one (defined here as infection with HIV), the mean conversion time to a positive serum antibody test following introduction of the virus is thought to be about 42 days, but may be longer since the test will not be positive until sufficient antibodies are present. Therefore, a patient infected with HIV may not show up as HIV positive even though there is risk of transmission of the virus during an exchange of, or contact with, infected body fluids. The HIV damages both the immune and the

neurologic systems. The principal routes of transmission are sexual, blood, and perinatal contacts. Common oral findings in patients with AIDS include candidiasis, herpes, hairy leukoplakia (figure 2-19), and Kaposi's sarcoma (figure 2-20).

Figure 2-19. Hairy leukoplakia on the lateral border of the tongue.

Figure 2-20. Kaposi's sarcoma on the dorsal surface of the tongue.

2-36. GONORRHEA

Normally a sexually-transmitted disease, gonorrhea is caused by the gram-negative diplococcus, *Neisseria gonorrhoea*. Primary infection is by genital contact and secondary infection is possible via the hands.

2-37. SYPHILIS

a. **General.** Syphilis is a sexually transmitted disease caused by a spirochete called *Treponema pallidum*. Oral lesions often are not present with syphilis. However, an active lesion or infective blood may transmit syphilis through a break in the skin or mucosal surface. All oral health providers should be knowledgeable concerning the infectious potential of syphilitic lesions and take necessary precautions (routine use of gloves during treatment, sterilization, and so forth). Syphilis can be classified as acquired and congenital.

b. **Primary Syphilis**. Acquired syphilis has three stages--primary, secondary, and tertiary. The first stage of syphilis is characterized by the appearance of a lesion called the chancre (primary lesion). The chancre is highly infectious and may appear anywhere on the body. Although it is commonly found on the genitalia, its appearance in the mouth is not rare (the site of possible oral-genital contact). A chancre in the oral cavity is characterized by a lesion with a hard, rolled border. Since it is usually painless, the patient is sometimes unaware of its existence. Usually, it is a solitary lesion. Any hard lesion appearing on the lips or in the mouth should be viewed with suspicion. The dentist and the dental specialist should never overlook the possibility of its presence and the attendant danger of infection. The primary lesion will heal spontaneously but, unless treated, will progress to secondary syphilis.

c. **Secondary Lesions**. The second stage of syphilis appears from 3 to 8 weeks after the appearance of the primary lesion. *Treponema pallidum* enter the bloodstream at the site of the primary lesion, multiply, and are carried throughout the body by the circulatory system. Consequently, the secondary lesions may appear anywhere on the body and in large numbers. The secondary lesions on the oral mucosa are known as mucous patches. The mucous patch is usually oval in shape with a moist, glistening, grayish-white, slightly-raised surface. Other secondary lesions may have a radically different appearance. This makes the clinical diagnosis of secondary syphilis difficult. **As with the primary lesion, mucous patches are highly contagious.** The dental officer and his assistants must be suspicious of any lesions of the mucous membranes of the mouth and throat and should obtain a laboratory diagnosis.

d. **Tertiary Syphilis**. The third and terminal stage of syphilis may appear at any time from several months to several years after the initial infection. The lesions of this stage, called gummas, are not very contagious. They rarely contain causative organisms. They appear in the mouth less frequently than secondary lesions. They normally appear as nodules and slowly get bigger, becoming softer and less dense. Eventually the center of the nodule becomes necrotic and a destructive lesion results. The soft tissue and underlying bone are destroyed. When gummas appear in the oral cavity, they often result in perforations of the soft palate.

e. **Congenital Syphilis**. A mother having syphilis during pregnancy may transmit the disease to the child. Certain tooth formations are considered typical of congenital syphilis if the child is not treated. In these cases, the incisors are notched on their incisal edges and shaped like a screwdriver. The cusps of the first molars are underdeveloped. The typically notched, screwdriver-shaped incisors are called Hutchinson's incisors. Deformed (mulberry) molars are another diagnostic sign. The presence of these teeth alone, however, should not be considered sufficient reason for making a diagnosis of congenital syphilis.

Continue with Exercises

EXERCISES, LESSON 2

INSTRUCTIONS. The following exercises are to be answered by marking the lettered response that best answers the question or best completes the incomplete statement or by writing the answer in the space provided.

After you have completed all of these items, turn to "Solutions to Exercises" at the end of the lesson and check your answers.

1. A surface tumor that may grow anywhere on oral mucosa and that grows outward like a cauliflower is a(n):

   a. Pyogenic granuloma.

   b. Papilloma.

   c. Odontoma.

   d. Fibroma.

2. Oral cancer may appear as:

   a. Leukoplakia.

   b. Erythroplakia.

   c. An ulceration.

   d. "a" and "b."

   e. "a," "b," or "c."

3. Cancer of the tongue tends to metastasize _____ in development.

   a. Early.

   b. Late.

4. A bright-red tongue and a burning sensation are characteristics of:

   a. Macroglossia.

   b. Ankyloglossia.

   c. Hairy tongue.

   d. Systemic smooth tongue.

5. Multiple or coalescing papillomas may indicate:

   a. Hypertrophied filiform papillae.

   b. A complex odontoma.

   c. Secondary syphilis.

   d. Squamous cell carcinoma.

   e. Partial ankyloglossia.

SPECIAL INSTRUCTIONS FOR EXERCISES 6 THROUGH 10. Match the name of the condition or disease in Column B to its chief symptom in Column A and write your answer in the space provided at the beginning of each exercise.

COLUMN A	COLUMN B
___ 6. A double crown is attached to a single root; a twin formation.	a. Geographic tongue
___ 7. The development of more teeth than the normal number.	b. Hypoplasia
___ 8. A tongue that is characterized by alternating red areas bordered by yellowish-white.	c. Mottled enamel
___ 9. Lack of development, or defective development, of the enamel of the tooth before its eruption.	d. Supernumerary teeth
___ 10. Form of enamel hypocalcification that results from the ingestion of excessive fluoride during enamel formation.	e. Gemination

SPECIAL INSTRUCTIONS FOR EXERCISES 11 THROUGH 15. Match the name of the condition or disease in Column B to its chief symptom in Column A and write your answer in the space provided at the beginning of each exercise.

COLUMN A	COLUMN B
___ 11. Ulcerative lesion associated with the first stage of syphilis.	a. Fibroma
___ 12. Disease in which the body is unable to use carbohydrates because of deficiency in insulin secretion of the pancreas.	b. Odontoma
___ 13. Notched, screwdriver-shaped incisors associated with congenital syphilis.	c. Chancre
___ 14. The most common benign tumor growth in the oral cavity.	d. Diabetes mellitus
___ 15. A benign tumor located within the facial bones or in the anterior or posterior dental arches, usually diagnosed from a radiograph.	e. Hutchinson's incisors

16. When tooth enamel is chalky, crumbles easily, and contains white or brown areas, the condition is known as:

    a. Concrescence.

    b. Enamel drops.

    c. Dens in dente.

    d. Hypocalcification.

    e. Anodontia.

17. Hemorrhaging, ulceration, spongy texture, magenta coloration, and enlargement of the gingiva are characteristics of:

    a. Leukemias.

    b. Hemophilia.

    c. Diabetes mellitus.

    d. Epilepsy.

    e. Pernicious anemia.

Check Your Answers on Next Page

SOLUTIONS TO EXERCISES, LESSON 2

1. b (para 2-3)
2. e (para 2-6)
3. a (para 2-6)
4. d (para 2-12)
5. c (figure 2-3 NOTE)
6. e (para 2-19d)
7. d (para 2-18)
8. a (para 2-10c)
9. b (para 2-14)
10. c (para 2-17)
11. c (para 2-37b)
12. d (para 2-28)
13. e (para 2-37e)
14. a (para 2-2)
15. b (para 2-5)
16. d (para 2-16)
17. a (para 2-27)

End of Lesson 2

LESSON ASSIGNMENT

LESSON 3	Fractures and Dislocation of the Jaw.
LESSON ASSIGNMENT	Paragraphs 3-1 through 3-6.
LESSON OBJECTIVES	After completing this lesson, you should be able to:

3-1. Identify the common signs and symptoms of fractures of the jaws.

3-2. Identify the common classification of fractures.

3-3. Identify fractures of the facial bones.

3-4. Identify guidelines for treatment of fractures.

3-5. Identify what action should be taken for a dislocated jaw.

SUGGESTION After studying the assignment, complete the exercises at the end of this lesson. These exercises will help you to achieve the lesson objectives.

LESSON 3

FRACTURES AND DISLOCATION OF THE JAW

3-1. GENERAL

Fractures and dislocation of the jaw occur frequently among members of the military population, particularly in combat. Immediate treatment involves lifesaving techniques to maintain breathing, control hemorrhage and shock, and observation for possible brain damage. Immediate treatment should also include immobilization of the head and neck to prevent damage to the spinal cord until the possibility of injury to the cervical spine has been ruled out at a definitive care facility. Among the more common traumatic facial injuries are fractures of the mandible and maxilla. Displaced bone segments from fractures of the maxilla and bilateral subcondylar or parasymphysis fracture of the mandible may result in airway problems. These fractures are often associated with soft tissue injury or loss, bone loss, and comminuted or impacted fragments of bone. Jaw fractures and associated injuries should be referred to the dental officer (usually an oral surgeon) for treatment. A diagnosis is usually established following a thorough examination that includes visual inspection, palpation, and radiographs.

3-2. COMMON SIGNS AND SYMPTOMS

Most patients with jaw fractures have a history of trauma and complain of pain. In addition, many patients have abnormal mobility of the fractured jaw and trismus (muscle spasm). Some common signs and symptoms of mandibular fractures include: malocclusion, laceration over the fracture site, ecchymosis (bleeding into the skin or mucosa) in the floor of the mouth, step defect, paresthesia (numbness or abnormal sensation), lack of condylar movement on opening, lateral deviation on opening, and the inability to open the mouth. Fractures of the maxilla and other bones of the mid-face have the following common signs and symptoms--distortion of facial symmetry, open bite because of displacement of the maxilla, ecchymosis, and paresthesia.

3-3. CLASSIFICATION OF FRACTURES

Fractures may be classified by their severity and tissue involvement. Figure 3-1 has examples of some types of fractures.

a. **Simple Fracture.** A simple fracture is a break in the bone that does not produce an open wound in the skin. A simple fracture can be complete (complete severance of the bone) or incomplete. Tissue adjacent to the fracture may or may not suffer considerable injury.

NOTE: A greenstick fracture is one in which one side of the bone is broken and the other side is bent.

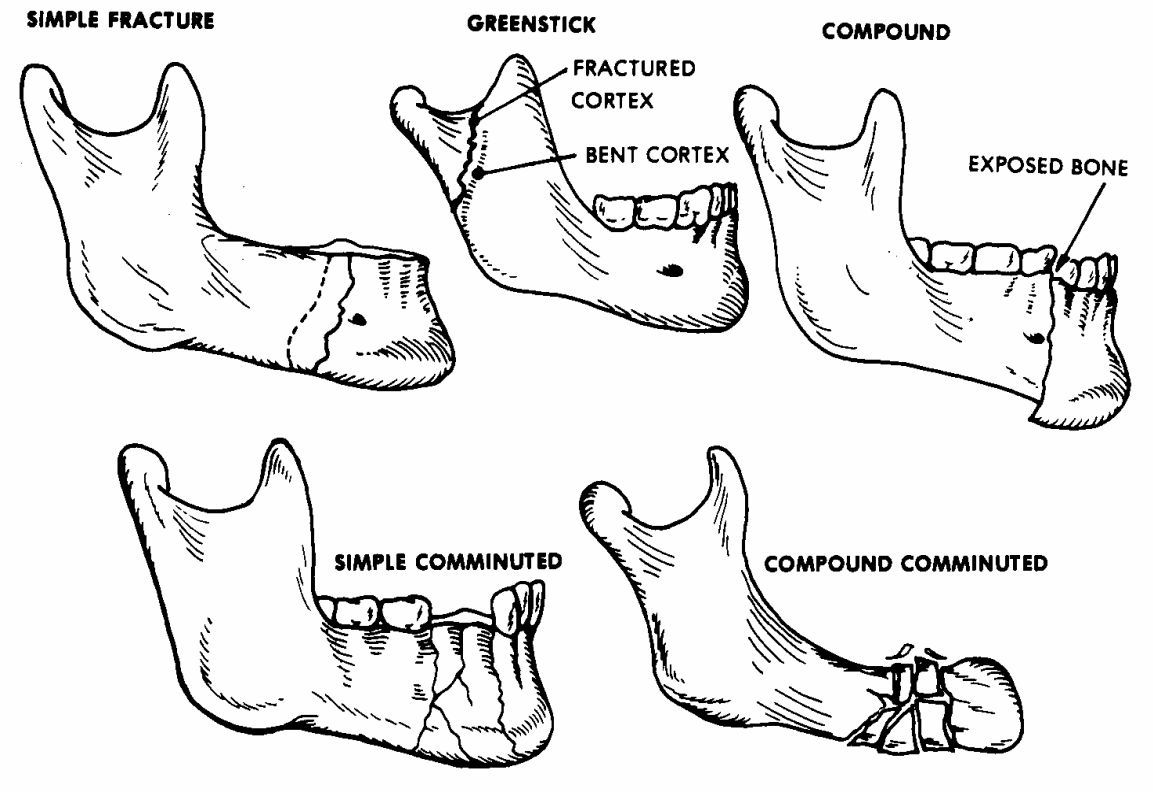


Figure 3-1. Types of mandibular factures.

b. **Compound Fracture.** A compound fracture is a break in the bone with an external wound extending to the bone. Communication from the bone to the skin or other covering surface is an invitation for contamination.

c. **Comminuted Fracture.** A comminuted fracture is one in which the bone is splintered into three or more fragments or is crushed.

NOTE: A compound-comminuted fracture is one with both a splintering of the bone and a break in the bone with an opening to the covering surface.

d. **Depressed Fracture.** A depressed fracture is a break in which the fractured part is driven below the normal level of the bone, as in a skull fracture.

e. **Impacted Fracture.** An impacted fracture is a b
ak in which the hard cortical bone of one fragment is driven into the softer cancellous bone of another fragment.

f. **Pathologic (Spontaneous) Fracture.** A pathologic fracture is a break without external violence at an area of the bone that has been weakened by a local disease.

g. **Multiple Fracture.** A multiple fracture is a break in which two or more fractures occur in the same or different bones.

h. **Favorable Fracture.** A favorable fracture is when the line of the fracture occurs in a direction that does not allow the pull of the muscles on the segments to displace the segments.

i. **Unfavorable Fracture.** An unfavorable fracture is a fracture with displacement or separation of the fractured segments because of muscle pull on the segments.

3-4. FRACTURES OF FACIAL BONES

a. **Fractures of the Maxilla.** Fractures of the maxilla commonly occur as horizontal fractures through the floor of the nose, horizontal fractures of the premaxillary area, tuberosity fractures, alveolar process fractures, and nasal process fractures. The nasal bone is the most common facial bone fracture. See figure 3-2.

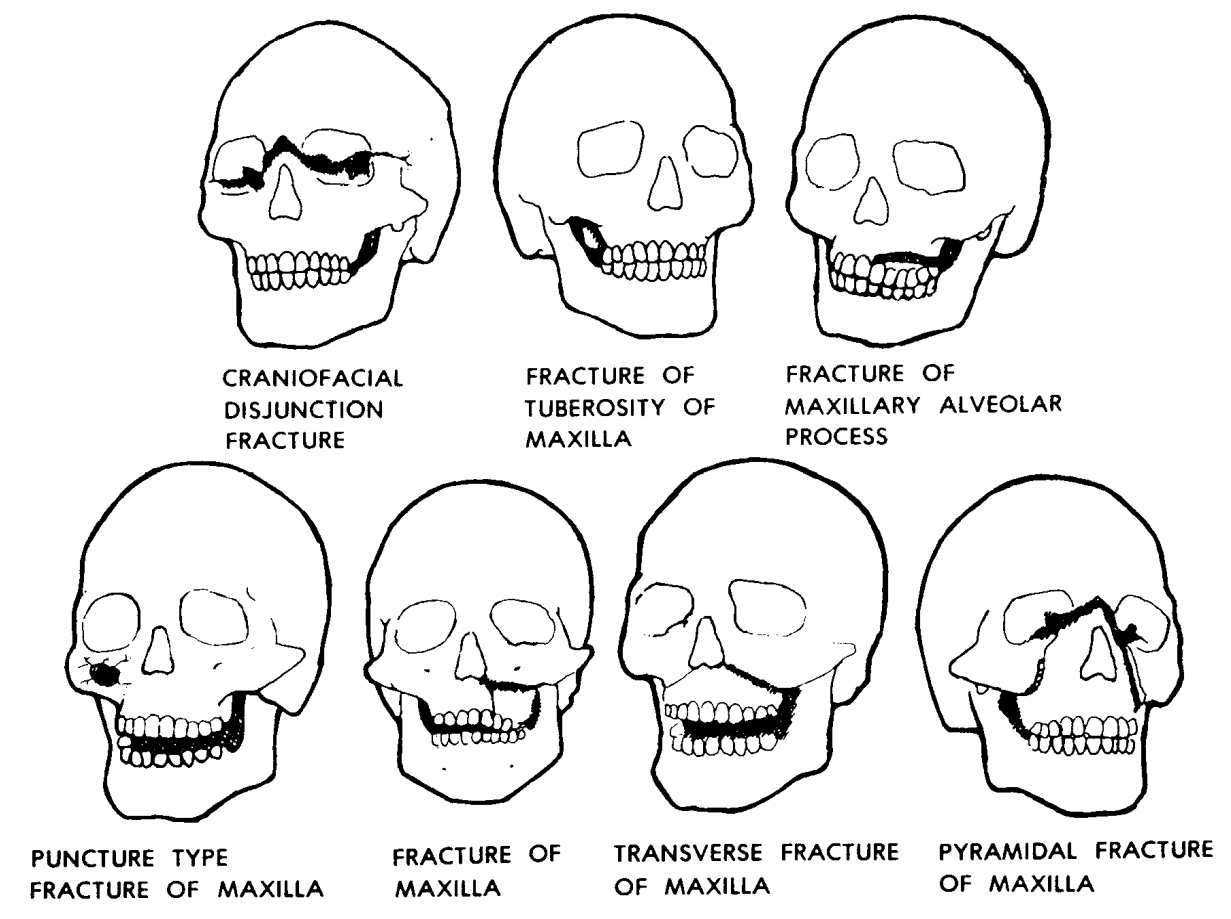

CRANIOFACIAL DISJUNCTION FRACTURE
FRACTURE OF TUBEROSITY OF MAXILLA
FRACTURE OF MAXILLARY ALVEOLAR PROCESS
PUNCTURE TYPE FRACTURE OF MAXILLA
FRACTURE OF MAXILLA
TRANSVERSE FRACTURE OF MAXILLA
PYRAMIDAL FRACTURE OF MAXILLA

Figure 3-2. Fractures of the maxilla.

b. **Fractures of the Mandible.** The mandible is the second most commonly fractured facial bone. Fractures of the mandible commonly occur in the body of the mandible, the neck of the condyle, and the angle of the mandible. See figure 3-3.

c. **Fractures of the Zygomatic Bone.** Fractures of the zygomatic bone include fractures involving the zygomatic arch (cheekbone) as well as the temporal, frontal, and maxillary bones. (See subcourse MD0501 for more information on the anatomy of the face.)

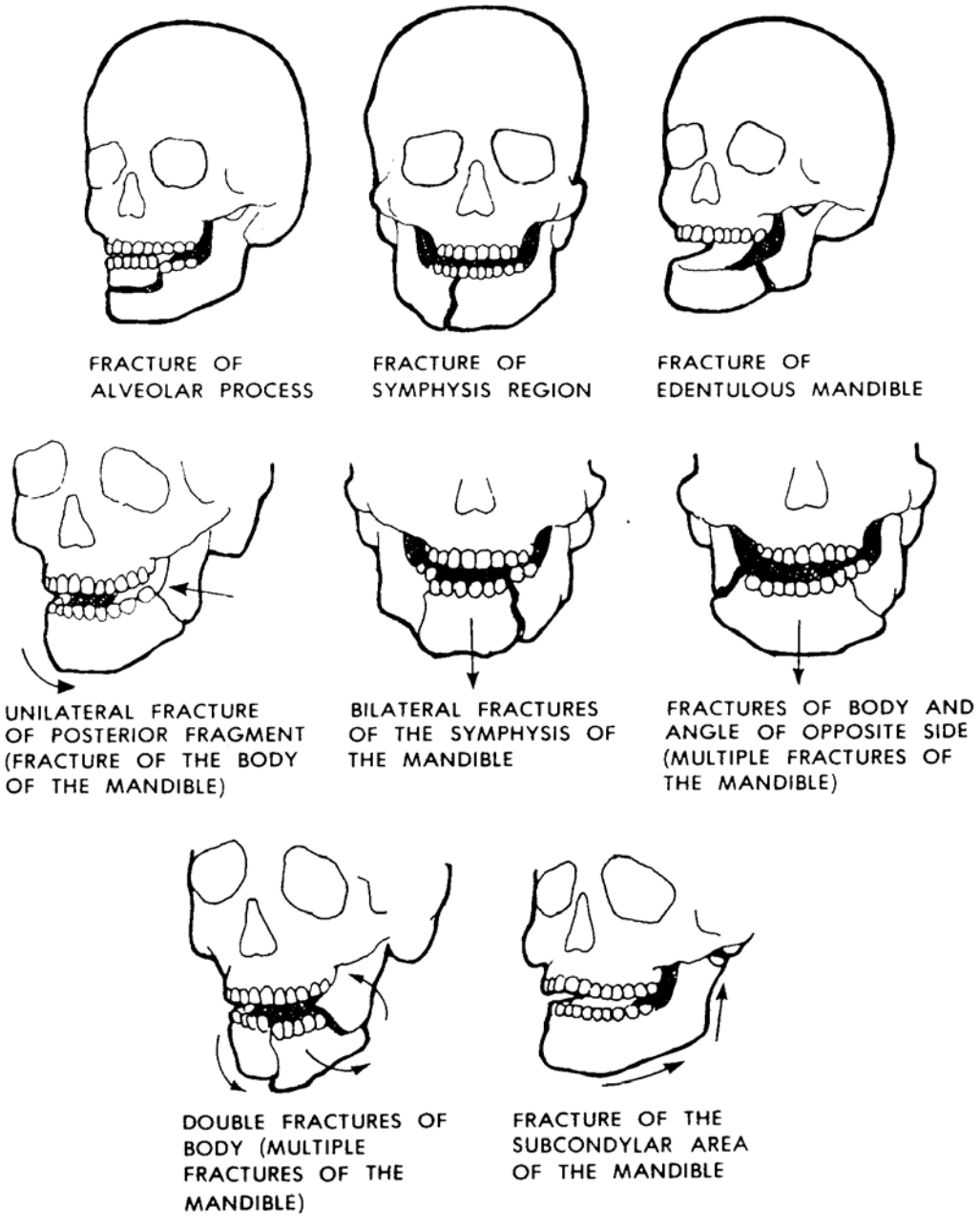

FRACTURE OF ALVEOLAR PROCESS
FRACTURE OF SYMPHYSIS REGION
FRACTURE OF EDENTULOUS MANDIBLE
UNILATERAL FRACTURE OF POSTERIOR FRAGMENT (FRACTURE OF THE BODY OF THE MANDIBLE)
BILATERAL FRACTURES OF THE SYMPHYSIS OF THE MANDIBLE
FRACTURES OF BODY AND ANGLE OF OPPOSITE SIDE (MULTIPLE FRACTURES OF THE MANDIBLE)
DOUBLE FRACTURES OF BODY (MULTIPLE FRACTURES OF THE MANDIBLE)
FRACTURE OF THE SUBCONDYLAR AREA OF THE MANDIBLE

Figure 3-3. Fractures of the mandible.

3-5. TREATMENT OF FRACTURES

Treatment of fractures requires restoration of the parts to their normal positions (reduction of a fracture) and immobilization (fixation) of the parts for about 6 to 8 weeks until a union between the bony parts takes place. Reduction of the fracture may be either closed or opened. In a closed reduction, the bone segments are manipulated back into position without surgically exposing the bone. Usually, arch bars and wires are used for fixation. In an opened reduction, the fractured bone segments are surgically exposed, which allows the fracture to be reduced exactly because of unobstructed, direct vision. In opened reductions, fixation generally is accomplished by drilling holes on either side of the fracture and using wire or metal plates to hold the segments in close approximation. Careful postoperative care is needed. Patient instruction in proper diet is essential because often the teeth are immobilized in the closed position, called intermaxillary fixation (the jaws being wired together). A dental liquid or soft diet is used because it is a high protein and high carbohydrate diet that provides the nutrients necessary for the healing of fractured bones.

3-6. DISLOCATION OF THE JAW

In a dislocation of the mandible, the head of the condyle is displaced from its normal relationship with the glenoid fossa. The condylar head slips down and out of the glenoid fossa and in front of the articular tubercle or eminence. The patient is unable to close his mouth. Often, there is pain, discomfort, and swelling. Dislocation of the jaws may be caused by a blow, yawning, laughing excessively, or otherwise opening the mouth too wide. In all cases, the dental officer or some other trained person should be summoned immediately. It is essential to restore the joint to its normal position as rapidly as possible. This is done by placing the thumbs in the posterior sulcus of the mandible in the region of the molar teeth and pressing downward and backward to slip the condyle under the articular tubercle. Since the jaw is likely to slip back into place quickly, it is essential to prevent the anterior teeth from being traumatically fractured.

Continue with Exercises

EXERCISES, LESSON 3

INSTRUCTIONS. The following exercises are to be answered by marking the lettered response that best answers the question or by completing the incomplete statement or by writing the answer in the space provided at the end of the question.

After you have completed all the exercises, turn to "Solutions to Exercises" at the end of the lesson and check your answers.

1. A fracture in which one side of the bone is broken and the other side is bent is a:

   a. Compound-comminuted fracture.

   b. Simple fracture.

   c. Greenstick fracture.

   d. Multiple fracture.

2. A fracture that occurs in a direction that does not allow the pull of the muscles on the fractured segments to displace the segments is:

   a. A favorable fracture.

   b. An unfavorable fracture.

3. Which of the following signs and symptoms of fractures is **NOT** a characteristic of mandibular fractures?

   a. Paresthesia.

   b. Ecchymosis.

   c. Malocclusion.

   d. Step defect.

   e. Nose bleed.

SPECIAL INSTRUCTIONS FOR EXERCISES 4 THROUGH 10. Match the classification (name) of the fracture in Column B to its description in Column A. Write your answer in the space provided.

COLUMN A		COLUMN B
___	4. Two or more fractures occurring in the same or different bones	a. Simple fracture
___	5. The hard cortical bone of one fragment is driven into the softer cancellous bone of another fragment	b. Compound fracture
___	6. The bone is broken and splintered into three or more fragments or is crushed	c. Comminuted fracture
___	7. A break in the bone that does not produce an open wound in the skin	d. Depressed fracture
___	8. A break without external violence at an area of the bone that has been weakened by a local disease.	e. Impacted fracture
___	9. The fractured part is driven below the normal level of the bone	f. Pathologic fracture
___	10. A break in the bone with an external wound extending to the bone.	g. Multiple fracture

11. Fixation is accomplished by drilling holes on either side of the fracture and using wire or metal plates to hold the segments in close approximation in:

    a. A closed reduction.

    b. An opened reduction.

12. It is necessary to immobilize a fracture of the jaw for _____ to _____ weeks until a union between the bony parts takes place.

13. When there is post-operative intermaxillary fixation, a dental liquid or soft diet is used that provides the nutrients necessary for the healing of fractured bones. This diet is a:

    a. High protein, high carbohydrate diet.

    b. Low protein, low carbohydrate diet.

    c. Low protein, high carbohydrate diet.

    d. High protein, low carbohydrate diet.

14. A soldier has a dislocated jaw. What action should be taken?

    Immediately look for a _____ person (such as a _____

    _____) to restore the joint to its normal position.

15. When trying to restore a dislocated jaw to its normal position, it is essential to

    prevent the _____ from being traumatically

    fractured.

16. The figure below illustrates:

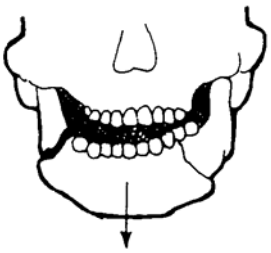
    a. Fractures of the edentulous mandible.

    b. A fracture of the subcondylar area of the mandible.

    c. A fracture of the body of the mandible.

    d. Fractures of the body and the angle of the opposite side.

    e. Fractures of the alveolar process.



17. The figure below illustrates a:

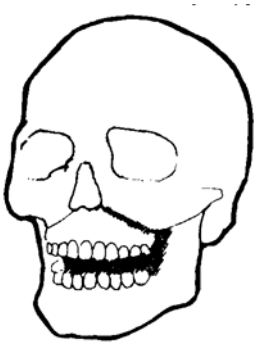
    a. Pyramidal fracture of the maxilla.

    b. Transverse fracture of the maxilla.

    c. Fracture of the maxillary alveolar process.

    d. Cranio-facial disjunction fracture.

    e. Fracture of the maxilla.



Check Your Answers on Next Page

SOLUTIONS TO EXERCISES, LESSON 3

1. c (para 3-3a NOTE)
2. a (para 3-3h)
3. e (para 3-2)
4. g (para 3-3g)
5. e (para 3-3e)
6. c (para 3-3c)
7. a (para 3-3a)
8. f (para 3-3f)
9. d (para 3-3d)
10. b (para 3-3d)
11. b (para 3-5)
12. six; eight (para 3-5)
13. a (para 3-5)
14. trained; dental officer (para 3-6)
15. anterior teeth (para 3-6)
16. d (figure 3-3)
17. b (figure 3-2)

End of Lesson 3